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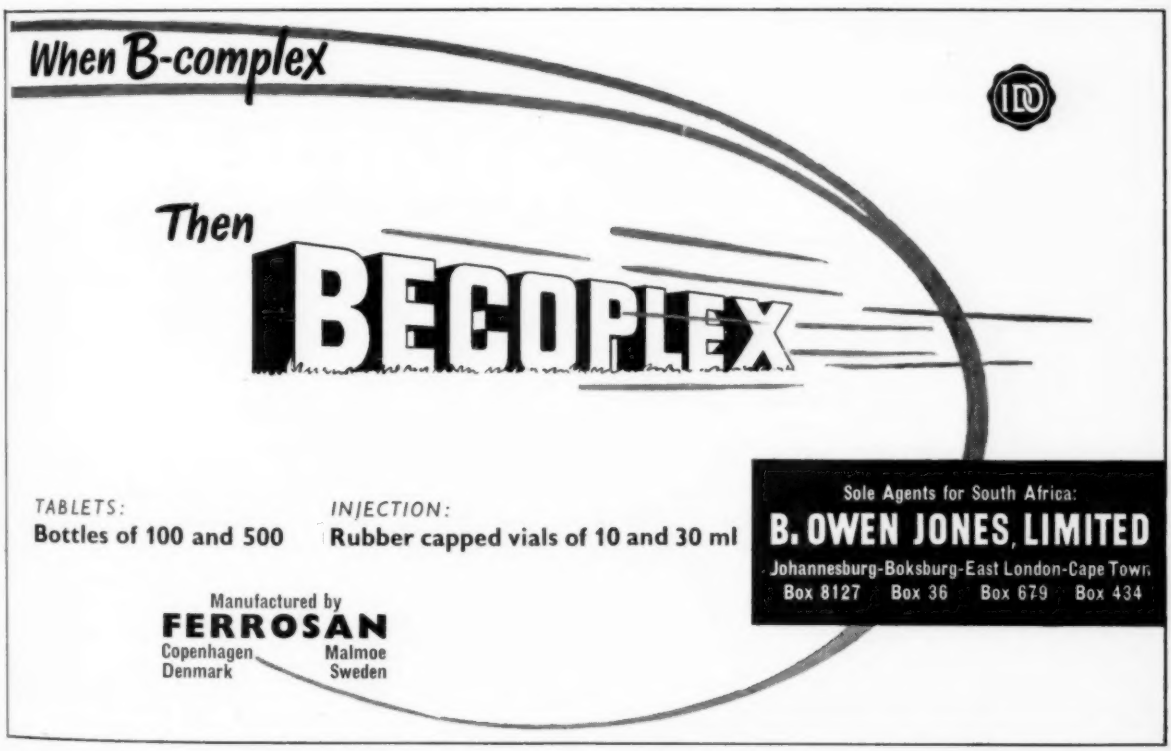

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EDITORIAL

CHLORDANE POISONING

The Council on Pharmacy and Chemistry of the American Medical Association performs many useful services for the medical and allied professions. Through its committee on pesticides information is made available from time to time on insecticidal agents of immediate medical interest. A recent addition to the series of reports of this nature is one dealing with chlordane,[1,2] a domestic and agricultural insecticide widely used in America.

The insecticidal properties of this chlorinated hydrocarbon were discovered in the United States and in Germany about the same time, but it was in America that chlordane was first made available—shortly after World War II. The compound is a heavy, dark brown, oily liquid soluble in the common organic solvents. The commercial product contains an admixture of 25-40% of related compounds consisting of isomers that have been separated by chromatography, and other substances of different chemical nature; these additional compounds contribute in some degree to the insecticidal action of the final product and also to its harmful effects.

The lethal action is achieved by contact, ingestion or exposure to the vapour of this viscous pungent agent. In man absorption can take place through the skin, the gastro-intestinal tract, or the respiratory tract; rapid penetration can occur, and the higher motor cortex and the cerebellum are then affected. The symptoms of acute chlordane poisoning are similar to those occurring with dicophane (chlorophenothane; DDT) and other chlorinated hydrocarbon insecticides. Irritability, salivation, dyspnoea, tremors and convulsions, followed by death, are the features that have been observed experimentally in animals, and also in human beings as the result of carelessness in handling the commercial mixture or from accidental ingestion or inhalation of chlordane. Other symptoms that have been reported in these cases are nausea, vomiting, diarrhoea, abdominal pain, cough, blurred vision, ataxia, confusion, delirium and mania.

Acute signs of poisoning may appear as early as

VAN DIE REDAKSIE

CHLOORDAAN-VERGIFTIGING

Die Raad vir Farmasie en Chemie van die American Medical Association lewer waardevolle dienste aan mediese en verwante professies. Die Raad se komitee vir plaagbestryding stel van tyd tot tyd verslae oor insekdodende middels beskikbaar wat van onmiddellike belang vir die mediese professie, is. Onlangs het 'n verslag verskyn oor chloordaan[1,2] 'n insektedoder wat algemeen in Amerika in die huis en op plase gebruik word.

Die insekdodende eienskappe van hierdie chloorkoolwaterstof is amper gelyktydig in die Verenigde State en Duitsland ontdek maar dit was in Amerika dat chloordaan die eerste keer beskikbaar gestel is—kort na Wêreldoorlog II. Dit is 'n swaar, donkerbruin, olierige vloeistof wat in gewone organiese oplosmiddels oplosbaar is. 25-40% van die handelsproduk is verwante verbindings (wat bestaan uit isomere wat chromatografies geskei is) en ander stowwe wat verskillend van chemiese aard is; hierdie addisionele mengsels verhoog tot 'n mate die insekdodende werking van die middel maar terselfdertyd ook die skadelike gevolge daarvan.

Kontak met, ingestie van of blootstelling aan die wasem van hierdie klewerige skerp middel kan noodlottig wees. Absorbering vind by die mens plaas deur die vel, in die maagdermkanaal en die lugweë; deurdringing kan snel geskied en dit tas dan die hoër motoriese skors en die cerebellum aan. Die simptome van akute chloordaanvergiftiging is soortgelyk as dié van dikofaanvergiftiging (kloorfenotaan, DDT) en ander chloorkoolwaterstof-insektedoders. Prikkelbaarheid, kwyling, kortasemigheid, bewing en stuiptrekkings gevolg deur die dood is kenmerke wat waargeneem is by eksperimente met diere en ook by die mens te wyte aan nalatigheid in die gebruik van die handelsartikel of as chloordaan per ongeluk ingeasem of ingeneem word. Ander simptome wat by hierdie gevalle aangetref word is mislikheid, vomering, diarree, maagpyn, hoes, dofsien, koördinasiesteuring, verwarring, en ylhoofdigheid.

Akute tekens van vergiftiging kan 45 minute na ingestie voorkom, en die dood mag binne 24 uur intree, maar dit kan ook daelank vertraag word na 'n enkel

45 minutes after ingestion, and death may occur within 24 hours but it may be delayed for many days after a single oral toxic dose. In fatal cases the dominant pathological signs that have been observed in human beings are generalized congestion, oedema, and signs of gastro-intestinal irritation.

In chronic chlordane poisoning symptoms indicate disturbances in the central nervous system and the optic nerve, while post-mortem examination has revealed degenerative cell-changes in the liver and kidneys.

A few deaths in human beings are already on record.[1] In one case a young woman died within a few minutes after spilling a suspension of chlordane on her dress; in another, death occurred 9½ days after suicidal ingestion of 6 g. of chlordane. Several instances of non-fatal poisoning have been noted. Contact dermatitis has occurred in a number of cases among exterminators and others handling this irritant poison.

In the treatment of poisoning the symptomatic measures used are the same as for other chlorinated hydrocarbon insecticides; removal of poison from the skin by *immediate* washing with soap and water or, if the chlordane has been swallowed, gastric lavage followed by a saline purgative. Milk and oils must be avoided. Barbiturates may be indicated when there is overstimulation of the central nervous system, but should be used with care.

The high toxicity of chlordane has militated against its use in the treatment of pediculosis and scabies, as compared with dicophane (DDT) and gamma benzene hexachloride. In its use for the control of agricultural and household pests it is dangerous to persons exposed to it. Even when applied to selected areas care is required to avoid deposits on surfaces played on by children. The slow liberation of fumes when it is applied indoors constitutes a danger. Its use on food crops is only permissible at certain stages of their development.

1. The Present Status of Chlordane (1955): J. Amer. Med. Assoc., **158**, 1364.
2. Fatal Chlordane Poisoning (1955): *Ibid.*, **158**, 1367.

mondelingse toksiese dosis. In noodlottige gevalle by die mens is die uitstaande patologiese tekens algemene kongestie, edeem en tekens van maagdermirritasie.

Simptome by kroniese chloordaanvergiftiging dui op stoornisse in die sentrale senuweestelsel en die gesigsenuwee, terwyl lykskouings degeneratiewe selveranderings in die lewer en niere aantoon.

'n Paar sterfgevalle is alreeds noteer.[1] In een geval het 'n jong vrou binne 'n paar minute beswyk nadat sy 'n chloordaan suspensie op haar rok uitgestort het; in 'n geval van selfmoord het die dood ingetree 9½ dae na die ingesie van 6 g. chloordaan. Verskeie gevalle van vergiftiging sonder noodlottige gevolge is aangeteken. In 'n aantal gevalle het huidontsteking (as gevolg van kontak) voorgekom by uitroeiers wat met hierdie prikkelgif werk.

Die simptomatiese behandeling is dieselfde as in die geval van ander chloorkoolwaterstof-insektedoders; verwyder die gif van die vel deur *onmiddellik* met seep en water te was. As chloordaan ingesluk is moet die maag gespoel word en 'n soutpurgasie toegedien word. Melk en olies moet vermy word. In geval van oorstimulasie van die sentrale senuweestelsel kan barbiturate nuttig wees maar hul moet in oorleg en met sorg gebruik word.

Te wyte aan die hoë toksisiteit van chloordaan moet dit nie vir die behandeling van pedekulose en skurfte gebruik word nie en is dikofaan (DDT) en gamma benseenheksachloried verkieslik.

Die gebruik daarvan in die huis en op die plase stel diegene wat daaraan blootgestel word in gevaar. Selfs as dit net in uitgesoekte gebiede gebruik word moet voorsorg getref word om te verhoed dat dit neerslaan op plekke waar kinders mag speel. Die stadige vrystelling van dampe binnenshuis is gevaarlik. Dit moet slegs vir voedselgewasse op sekere stadia van hul groei gebruik word.

1. The Present Status of Chlordane (1955): J. Amer. Med. Assoc., **158**, 1364.
2. Fatal Chlordane Poisoning (1955): *Ibid.*, **158**, 1367.

'VANISHING TOES'

A *Lancet* annotation[1] makes reference to the case which Dr. D. M. Krikler[2] recently reported in the *South African Medical Journal* in an article entitled 'The Case of the Vanishing Toes'. The writer of the annotation agrees with Dr. Krikler that the case is probably an example of 'idiopathic osteolysis'. The patient (seen in January 1955) was a 35-year-old Coloured male, in whom, 5 years before, certain toes (the 1st and 4th of the right foot and the 4th and 5th of the left) had disappeared wholly, or almost wholly, in the course of a few months without any history of pain or trauma or exposure to cold.

On X-ray examination, all the phalangeal bones of the missing toes were seen to have disappeared, except a small piece of the proximal phalangeal bone of two of them; and the distal end of one of the metacarpals was also missing and there were signs of erosion of the distal phalanges of two of the remaining toes. The lesion consisted of erosion with no evidence of reaction. Further, there was fusion of the cuneiforms and synostosis of the 4th and 5th metatarsals on the right; the evidence suggested that these last conditions were congenital. X-ray revealed no other lesions in the rest of the skeletal system (apart from the absence of the right arm, which was missing from the level of the shoulder joint; the patient said that the loss had occurred in childhood, when he was mauled by a tiger in a circus); and the history and examination revealed nothing suggesting a cause for the loss of the toes. The patient was a 'steady' drinker and malnourished, with hepatomegaly and pellagrinous changes in the arms and the dorsa of the feet.

Gorlam *et al.*[3] were only able to find 16 cases of disappearing bones reported in the literature. They discount trauma as a possible cause, and state that biopsies indicate that a common feature is an over-

growth of thin-walled blood-vessels, and this haemangiomatosis may be an aetiological factor. Progress of the condition is slow and variable; it commonly stops after a time but occasionally ends fatally.

1. Annotation (1956): Lancet, **1**, 93.
2. Krikler, D. M. (1955): S. Afr. Med. J., **29**, 1050.
3. Gorham, L. W., Wright, A. W., Shultz, H. H. and Maxon, F. C. jun. (1954): Amer. J. Med., **17**, 674

SURGERY OF THE GALL-BLADDER AND THE COMMON BILE-DUCT*

C. A. R. SCHULENBURG, M.D., M.CH., F.R.C.S.
Pretoria

I. SURGERY OF THE GALL-BLADDER

The surgical treatment of diseases of the gall-bladder consists largely of the treatment of stones and their complications. There is a voluminous literature on gallstones, but its analysis would serve little purpose in this paper. It is of greater value to analyse one's own experience, to discuss how we can diagnose lesions of the biliary tract earlier, with greater accuracy, and how we can treat our patients more efficiently and with greater safety.

Cholecystectomy is one of the commonest of abdominal operations, usually associated with low mortality and morbidity; but this very fact has led to the mistaken view that it is also a simple operation. This light-hearted attitude is to be deplored for, not only may removal of the gall-bladder be an extremely difficult and dangerous operation, but it may be attended with the most serious accidents, such as damage to the common bile-duct.

I present for analysis a consecutive series of 250 cases (260 operations) of surgery of the biliary tract in private practice. This figure could have been more than doubled if one had included free cases operated on in hospital but, because clerking facilities are limited hospital cases are largely lost to follow-up. But although the analysis is confined to the more limited series the opinions expressed in this paper are based on the total experience.

This series of 250, being consecutive cases, is of more value than a selected group of cases of, say, calculous disease only, as it shows a fair cross-section of the type of biliary surgery which may be encountered in practice.

Table I shows an analysis of the type of biliary surgery performed in this series.

TABLE I

Cholecystectomy	230
Cholecystostomy	10
Stricture of common duct	3
Cholecysto-jejunostomy	2
Cystic duct, remnant excision	4
Secondary choledochostomy	4
Laparotomy, infective hepatitis	3
Carcinoma of gall-bladder, biopsy	1
Gallstone ileus	1
Sphincterotomy	2
Total operations	260

* Papers presented at the South African Medical Congress, Pretoria, 1955.

Fig. 1 indicates the age incidence in 230 cases of cholecystectomy. There were 160 females and 70 males. In both sexes the highest incidence occured in the 5th decade.

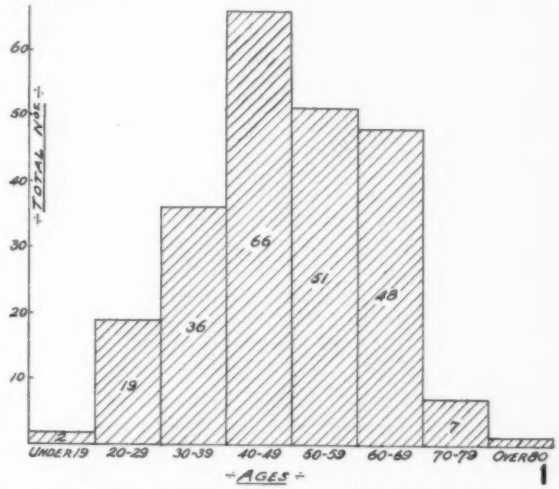

Fig. 1. Age incidence in 230 cases of cholecystestomy.

Table II shows an important analysis of the presence or absence of stones in the common bile-duct. This aspect of surgery of the biliary tract is discussed in Part II. The table indicates that, in all cases of cholecystectomy, it was considered necessary to explore the common duct in 29·1% of cases, that stones were present in 14·8% of all cases, and that, of all common ducts opened, stones were present in 50·7%.

TABLE II. STONES IN THE COMMON DUCT

Cases	*Common duct opened*		*Stones present*		*Common duct opened, found to contain stones*
	No.	*%*	*No.*	*%*	
230	67	29·1%	34	14·8%	50·7%

Table III analyses mortality rate. In cholecystectomy only, the mortality was *nil*. Where the common duct was explored, the mortality rate was 8·9%, making an average mortality rate in all cases of cholecystectomy,

with or without exploration of the common duct, of 2·6%.

TABLE III. OPERATIVE DEATHS (DEATHS DURING STAY IN HOSPITAL)

	Cases	*Deaths*	*Percentage Mortality*
Cholecystectomy only ..	163	0	0% } 2·6%
Cholecystectomy and/or Choledochostomy	67	6	8·9% }
Miscellaneous	20	3	15%
Total	250	9	3·6%

The high mortality rate of 8·9% in exploration of the common duct needs explanation, and details of the 6 deaths are given:

One was a case of carcinoma of the common duct, with long-standing jaundice. He died in hepatic coma 2 days after operation.

One case with hypertension died of cerebral haemorrhage 10 days after operation.

Two cases of impacted stone with 3 weeks' neglected jaundice died in hepatic coma a few days after operation. (One was a robust young parson, who had been under treatment by quacks and faith-healers for severe and deepening jaundice before coming to operation.)

One case of cholecystectomy and removal of stone in the common duct was a frail old man who burst his abdomen on the 10th day and died soon after re-suture.

One case was a frail old lady of 83, who died after difficult transduodenal removal of impacted stone.

The 3 deaths in the series of 20 cases of miscellaneous conditions were a case of carcinoma of the gall-bladder, a case of carcinoma of the stomach associated with acute cholecystitis in a man of 81, and a case of stricture of the common duct.

The mortality rate in the entire series of 250 cases (260 operations) was 3·6%.

FOLLOW-UP AND RESULTS

In this country it is hardly practicable for all patients to report personally for follow-up examination. Accordingly, follow-up by letter is resorted to. I believe this to have been valuable in the present investigation, for the patient is aware of his (or her) symptoms, and can state accurately whether pre-operative symptoms have gone, are still present, or have become aggravated. The patients were asked to reply to the following 4 simple questions:

1. Have you been quite well since the operation?
2. Have you had trouble with the bile passages?
3. Have you had a further operation for this trouble?
4. Any further comments?

If a patient reported further severe trouble, a personal letter was sent asking for details. In some cases further investigation was arranged.

Of the 250 cases, 229 were traced (a 92% follow-up). Ten cases had died since their discharge from hospital, from coronary thrombosis (5 cases), carcinoma of the pancreas (3 cases), aplastic anaemia (1 case) and periarteritis nodosa (1 case).

What we were interested in was the results of cholecystectomy. It is to be noted that, of the 230 cholecystectomies, in 35 (15%) the gall-bladder did not contain stones. In some cases operated on, the gall-bladder wall was found to be opaque and thickened, inflammation or cholesterosis being present but no calculi; in the absence of other pathology, the gall-bladder was removed. In a few cases, one was left with the real problem of a patient with classical biliary colic, in whom no pathology could be found on laparotomy. Should a normal-looking gall-bladder be removed? We have left the gall-bladder in some such cases, and the patient's pain has continued. In others we have removed the gall-bladder, and some patients have been relieved and others not.

Table IV indicates the results of cholecystectomy. (Note: 'Mild trouble' means insignificant and mild symptoms described as 'windy', 'have to take salts', 'dyspepsia', 'have to watch my diet', etc. 'Severe trouble' means severe and incapacitating symptoms, such as attacks of pain, and cases where the pre-operative symptoms are unchanged.)

TABLE IV. RESULTS OF CHOLECYSTECTOMY
(Average follow-up interval 3·5 years)

Cases	*Deaths*	*Survivors traced*	*No further trouble*	*Further trouble*	
				Mild	*Severe*
230 ..	8	196 (85%)	127 (65%)	44 (22%)	25 (13·7%)

It is noteworthy that in 13·7% of cases the patient had little or no relief from the operation.

Table V shows the results of cholecystectomy in cases with stones and without stones respectively. It is significant that, if 'mild' and 'severe' troubles are combined, more than 50% of cases of cholecystectomy without stones had further symptoms after operation.

TABLE V. RESULTS OF CHOLECYSTECTOMY

	Cases	*No further trouble*	*Further trouble*	
			Mild	*Severe*
G.B. contains stones	167	113 (67·6%)	33 (19%)	21 (12·5%)
G.B. does not contain stones ..	29	14 (48%)	11 (38%)	4 (13·7%)

Consideration of Table V brings us again to the problem of the patient with 'biliary colic', with the abdomen opened, no stones discovered, and the gall-bladder looking normal. It is our duty to search thoroughly for other pathology before removing a non-calculous gall-bladder. We believe that the state of the sphincter of Oddi should not be overlooked, for a 'fibrotic' state may be present. It is in this very type of case of persistent pain following removal of a non-calculous gall-bladder, that subsequent sphincterotomy has given good results.

Table VI shows results compared in cases where the common duct was or was not explored.

TABLE VI. RESULTS OF CHOLECYSTECTOMY

	Cases	*No further trouble*	*Further trouble*	
			Mild	*Severe*
Common duct not explored	145	92 (63·4%)	32 (22%)	21 (14·4%)
Common duct explored	51	35 (68·6%)	12 (23·5%)	4 (7·8%)

Significant trouble was experienced in twice as many cases where the common duct was not explored as in those where it was opened. This may indicate, of course, that stones in the common duct may have been overlooked in the first group of cases, and that we should extend our indications for exploring the duct. This has been difficult to establish, but it is possible that stones are present in the common duct in 4 of the 25 cases who experienced 'severe trouble' post-operatively. These may have been present at the time of the cholecystectomy, or they may have developed subsequently.

Consideration of the 25 cases with 'severe trouble', reveals the following (Table VII).

TABLE VII

	Cases
Common-duct stone probable *	4
Duodenal ulcer or gastric ulcer	2
Chronic relapsing pancreatitis	1
Probable chronic relapsing pancreatitis	2
Plummer-Vinson syndrome	1
Investigated with Biligrafin and barium meal—negative findings	3
Re-operated, sphincterotomy	1
Associated gastrectomy at primary operation †	2
One attack of pain without jaundice 5 years after primary operation	1
Attacks of 'fainting'	1
Unexplained and not investigated	7
Total	25

* In none of these cases was the common duct explored at the primary operation.
† Could account for symptoms.

Consideration of the 25 cases who did poorly after operation therefore reveals that at least 13 cases suffered from pathological conditions more or less unassociated with the original trouble or the operation. The case in which sphincterotomy was performed was completely relieved by this operation. In the 4 cases suspected of having common-duct stones, the duct was not explored originally because there appeared to be no indication for the procedure. Together with the 7 unexamined cases, there therefore remain 11 cases who may be said not to have benefited from their cholecystectomy.

GENERAL DISCUSSION

1. *Procedure*

Our procedure at an operation for cholecystectomy is as follows:

A high, substantial, right paramedian, muscle-displacing incision is made. This is adequate for all types of biliary surgery, provided it extends really high, right up to the costo-xiphoid junction. The abdominal wall is treated with the greatest gentleness, and a self-retaining retractor is never used; nor is a 'gall-bladder rest' or a sandbag behind the patient ever used.

A thorough and systematic examination is made of all abdominal and pelvic organs, including structures such as the oesophageal hiatus. (This procedure is indeed followed at every laparotomy, except in the presence of acute suppurative conditions.) If any additional pathological condition is found, it is usually dealt with at the same operation if the patient's condition allows. Gastrectomy, splenectomy, hiatus hernia, may all be done at the same time. The appendix is removed as a routine measure, unless it is completely inaccessible.

The peritoneum to the right of the duodenum is incised, and the duodenum, together with the head of the pancreas, is mobilized, so that the common duct and the pancreas can be properly palpated between the fingers and thumb.

Cholecystectomy is proceeded with in the usual manner from the neck upwards, after completely identifying the bile-ducts and the hepatic and cystic arteries. If there is even the slightest doubt about the anatomy around this area, the gall-bladder is removed from the fundus downwards. The cystic artery is ligated and divided, and care is taken to remove the entire cystic duct. The cystic duct is left if it would appear hazardous to proceed with its identification and removal; such cases are exceptional.

There is no opportunity in this paper to discuss the hazards and difficulties of this operation. It can be the easiest and quite the most difficult of abdominal operations. One should be acutely aware of the anatomical abnormalities of the region, the operation should proceed with the greatest care and without hurry, and no structure should be divided or ligated unless it has been absolutely identified.

At the conclusion of the operation, the gall-bladder bed is re-peritonealized, and the peritoneum is reconstructed over the common duct. A soft rubber drain of the Penrose type is used in all cases, and is brought up between the two lobes of the liver, through the upper part of the wound.

2. *Acute Cholecystitis*

Each case of acute cholecystitis is assessed individually and treated by antibiotics and replacement of fluid and electrolytes; and cholecystectomy is performed as soon as the general condition of the patient allows, usually within 1-3 days. In the aged, the acutely ill and the toxic patient, however, we still perform a cholecystostomy as a primary operation, frequently under local analgesia.

3. '*Silent Stones*'

Whenever a gall-stone is discovered, either during routine radiological investigation, or at laparotomy for other diseases, cholecystectomy is advised, either at the time or subsequently.

4. '*Post-Cholecystectomy Syndrome*'

There is no opportunity to discuss this aspect in detail. We believe that in all cases of post-cholecystectomy pain an organic cause must be sought, and in the majority of cases it will be found. 'Biliary dyskinesia' is a questionable diagnosis, attributed to functional spasm of the sphincter of Oddi. The following causes of post-cholecystectomy pain must be carefully considered:

1. *Errors in diagnosis.* Hiatus hernia, chronic pancreatitis, right kidney disease, duodenal ulcer, and coronary disease, should all be excluded pre-operatively, and also at laparotomy. We have found that posterior penetrating duodenal ulcer in women may give rise to intermittent attacks of pain, quite unlike the usual ulcer

history. It is also well to bear in mind that, should one operate for gall-bladder pathology and find none, a posterior duodenal ulcer may not easily be seen or felt unless one deliberately enters the lesser sac through one of the omenta, to examine the posterior aspect of the first part of the duodenum.

It should be stressed that a careful clinical evaluation of the patient is most important, with laboratory and X-ray studies as secondary aids in diagnosis.

2. *Errors in surgical technique.* It is a fault to leave the cystic duct behind. It is even worse to leave a portion of the gall-bladder behind unless its mucosa is destroyed. Such remnants, with or without stone, may give rise to quite marked symptoms, often entirely relieved by subsequent removal. Failure to remove stones from the common duct is often the cause of persistent symptoms, as is failure to perform a sphincterotomy when the sphincter is fibrotic and cannot be dilated.

The most serious error in technique is trauma to the common duct, and post-operative symptoms and signs are proportional to the extent of the damage.

3. *Residual disease.* Residual common-duct stones as well as untreated peptic ulcer, hiatus hernia, or chronic pancreatitis, may cause persistent symptoms after cholecystectomy. In the series of cases reported here, a most careful search was made for other conditions at the time of the cholecystectomy, and they were dealt with if discovered; yet 13·7% of the cases suffered from what may be classified as severe or significant 'post-cholecystectomy syndrome'. In one case pancreatitis was present at the time of the operation, and the patient has continued to have symptoms. In 2 cases peptic ulcer developed which was not present at the primary operation. In at least 11 other cases there were severe symptoms, usually consisting of the same attacks as before the cholecystectomy. In the absence of jaundice, common-duct stones are unlikely but cannot be excluded. Fibrosis or spasm of the sphincter of Oddi is a possibility.

One should consider, in the first place, extending the indications for exploring the common duct at cholecystectomy, so as to minimize the risk of leaving common-duct stones behind; and, secondly, investigating the state of the sphincter of Oddi more frequently and thoroughly, so that a sphincterotomy may, if necessary, be performed at the primary operation.

SUMMARY

1. A consecutive series of 250 cases (260 operations) of biliary-tract surgery in private practice is analysed. Emphasis is laid on the incidence of common-duct stones.

2. A complete follow-up study was undertaken, and the results of cholecystectomy are analysed.

3. The procedure of cholecystectomy is discussed.

4. 'Post-cholecystectomy syndrome' is discussed briefly.

SAMEVATTING

1. 'n Aaneenlopende reeks van 250 gevalle van operasies aan die galweë word (260 operasies) ontleed, met spesiale verwysing na stene in die ductus choledochus.

2. Die gevalle van cholesistektomie word ontleed en die resultate, verkry van 'n opvolgstudie, word uiteengesit.

3. Die operasie van galblaasverwydering word bespreek.

4. ‚Na-cholesistektomie sindroom' word bespreek.

II. SURGERY OF THE COMMON BILE-DUCT

The commonest condition of the common bile-duct which merits discussion is stones. We have to make a pre-operative diagnosis of stones in the common duct if at all possible. If stones are suspected, or if they have to be excluded, we should know how to search for them at operation. We should recognize certain indications for incising and exploring the common duct and, once the duct has been opened, we should know exactly what to do and how to avoid missing stones.

The very fact that there is a voluminous literature on the question of stones in the common duct overlooked at the time of cholecystectomy indicates the great importance of the problem; any procedure or technique, however trivial it may seem, for detecting and removing common-duct stones, should be familiar to the surgeon.

CLINICAL DIAGNOSIS

Stone v. Compression. The typical clinical picture of attacks of biliary colic, followed by shorter or longer periods of jaundice, in the case of stone, is well known. The more cases one sees, however, the more atypical syndromes are seen. There is no doubt that a case of insidious onset of painless jaundice, gradually deepening, may be due to an impacted stone. On the other hand, we have had cases of intermittent attacks of jaundice, sometimes accompanied by pain, proved at operation to be due to carcinoma of the pancreas, or chronic pancreatitis. It is well to remember that the great majority of cases of pancreas carcinoma have pain.

In our experience, Courvoisier's law has been so often proved wrong that it is of no practical importance, and could practically be disregarded in the differential diagnosis of jaundice.

Infective Hepatitis

A most difficult differentiation has often to be made between stone in the common duct and infective hepatitis. Clinically, hepatitis is usually ushered in by a period of 'feeling off-colour' for 7-10 days, the gradual onset of jaundice, enlarged tender liver, and absence of previous attacks of biliary colic. If the patient is young, this would favour hepatitis, but the elderly are more likely to have stones. However, one of our cases of hepatitis was 59 years of age.

Naturally we turn to the laboratory for help in the differential diagnosis of jaundice, and here we have had our biggest disappointments. Generally speaking, we

have found that laboratory tests might confirm a diagnosis which was clinically quite definite, but in cases where the diagnosis was doubtful the laboratory tests have been not helpful and, in some cases, misleading.

Severe liver damage early in the illness may point to hepatitis but, when obstructive jaundice caused by stone has been present for some time, similar liver damage may be demonstrated. Generally speaking, a high alkaline phosphatase points to obstructive jaundice, but even this has often not been helpful. We have had the mortification of attending a woman of 70 who had been diagnosed and treated as a case of hepatitis for 6 weeks and reached our department *in extremis*, at whose autopsy a large stone was found impacted in the common duct.

Mrs. H., aged 59, gave a history of abdominal discomfort and jaundice. She was seen by a physician who, supported by laboratory tests, made a diagnosis of hepatitis. When she did not improve on treatment, she resorted to quacks for 7 months. She came to us with severe liver damage, loss of weight, and deep jaundice. At operation, a large stone was removed from the common duct. She recovered well and is in good health.

Mr. D., aged 59, was admitted to the physicians with a history of lassitude, vague pain in the right upper quadrant, fever, and jaundice. The clinical diagnosis was hepatitis, but the laboratory report was an unequivocal one of obstructive jaundice. Because of the great uncertainty, operation was performed, and this revealed a normal gall-bladder, normal common duct, and an enlarged liver. The common duct was opened (to satisfy the pathologist), but there was no stone and no obstruction.

Mrs. B., aged 46, presented with a history of long-standing dyspepsia, with a recent attack of R.U.Q. pain, followed by jaundice. Blood examination showed an alkaline phosphatase of 11·25 K.A., and the pathologist thought that the case was not obstructive jaundice. At operation, gall-stones were found, with stone in the common duct.

Naturally, each case will be analysed and judged individually but, in case of great doubt, we feel it is safer to perform laparotomy to establish the diagnosis early on in the illness. It goes without saying that the anaesthetist will be consulted and will administer an anaesthetic non-toxic to the liver and kidneys and, if a diagnosis of hepatitis is confirmed, the operation will be completed as speedily as possible.

RADIOGRAPHY

In the diagnosis of stones, with a functioning or non-functioning gall-bladder, we have been very satisfied with the accuracy of oral cholecystography by Telepaque. For the demonstration of disease in the common duct, intravenous cholangiography by Biligrafin has been used. In our experience, this investigation is valuable but has its limitations. The shadow cast by the dye is frequently not very dense, and it is difficult to detect filling defects. We therefore say that when we have positive findings, such as a hugely dilated duct, or obvious filling defect, the investigation has helped us. If, however, the common duct shows up, but no obvious stones are seen, the presence of stones or other disease has not been excluded. The density of excreted dye also gives an indication of liver function. The investigation will thus also be of little use for the detection of stones in the presence of liver damage, for the excretion will be poor.

OPERATION

When an operation is performed for gall-stones, under what circumstances should the common duct be opened? Some surgeons solve this problem simply by making it a routine to incise and explore the common duct in every case of cholecystectomy. Others use the less satisfactory method of exploring by a bougie through the stump of the cystic duct. We do not think the former policy is wrong, *provided* the common duct is opened with great care, and explored with great gentleness. A common duct which is not dilated has thin walls and a narrow lumen and does not lend itself to easy exploration, so that some damage may be caused at the site of incision. A traumatic stricture may follow, which would be a great tragedy since the duct was normal in the first place. We therefore list the following indications for incising and exploring the common duct for stones:

1. Presence of jaundice, or history of jaundice. It is well to remember, however, that stones may be present in the common duct without jaundice having been present.

TABLE I

History of Jaundice	*Stones in Common Duct*	*No.*	*Percentage*
No	Yes	20	29·8%
No	No	18	26·8%
Yes	Yes	14	20·8%
Yes	No	15	22·4%

Table I indicates findings in a personal series of 230 cases of cholecystectomy, in which the common duct was explored in 67. Note that in 29·8% of cases stones were present in the common duct in the absence of history of jaundice.

2. Dilatation and/or thickening of the wall of the common duct. It is uncommon to find stones in a duct which is not dilated to a certain degree.

3. Small stones or 'gravel' in the gall-bladder. It is our routine to have a nurse open the gall-bladder immediately on removal so that its contents may be inspected. Small stones and gravel easily find their way into the common duct, especially when the cystic duct is wide.

4. Positive or suspicious findings on palpation of the common duct or head of the pancreas, or on operative cholangiography.

5. Presence of acute or chronic pancreatitis.

6. 'Peanut' gall-bladder—a small contracted gall-bladder indicates long-standing calculous disease, usually accompanied by dilated common duct containing stones.

7. Non-calculous gall-bladder in a patient with typical biliary colic. The state of the sphincter of Oddi has to be established.

Table II in Part I (page 183) indicates the findings in a series of 230 cholecystectomies performed by the author.

When the common duct has been opened

Once the common duct has been opened, a definite routine should be followed in a systematic search for stones. The opening in the duct should be made large enough to admit graduated bougies. The character of

the escaping bile should be noted, and gravel or stones removed. Palpation of the duct may reveal stones, which may be 'milked' to the opening. A urethral bougie is now gently inserted; palpation around the bougie is the surest method of detecting stones.

The usual type of gall-stone forceps for grasping a stone (Desjardin's) is not very satisfactory for use in the common duct, for it has not the correct angle. We use

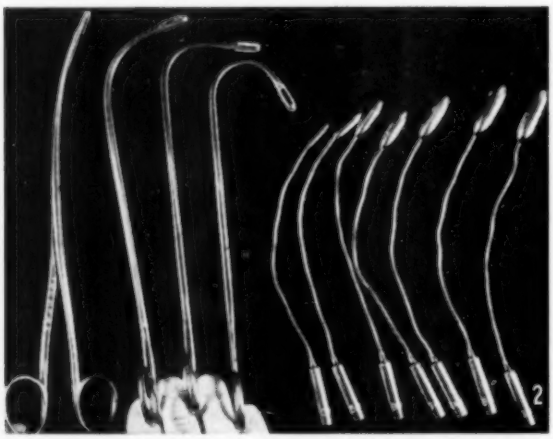

Fig. 2. Forceps for grasping stone in the common duct (left) and Bake dilators (right).

kidney stone forceps, which are bent to a right angle or more (Fig. 2); the ampulla of Vater is then dilated with Bake's dilators (Fig. 2)—it is usually not necessary to dilate to more than 7 or 8 mm. The dilators may be guided through the ampulla by grasping the head of the pancreas and the second part of the duodenum between fingers and thumb of the left hand. This can only be done if these structures are mobilized by incising the peritoneum lateral to the duodenum (this is done in every case of cholecystectomy so that the duct may be properly palpated). Now the duct is thoroughly flushed out with saline.

After proceeding in the manner outlined above, it is possible to be reasonably certain that no stones remain in the distal part of the common duct. It is a real problem, however, to know whether residual stones remain in the common hepatic duct, or in the intra-hepatic portions of the duct. Such stones may or may not be detected by palpation with bougies. We have found an excellent way of dealing with such stones: A large bulb syringe, or a bladder syringe, with blunt end, is inserted into the opening in the duct, and directed upwards. Saline is injected carefully, but under reasonable pressure, so as to fill the hepatic ducts and distend them. Stones become loosened and, if the syringe is now suddenly removed, the saline flushes back rapidly and stones are brought down. In one patient, in whom already 22 stones had been removed from the common duct and we had used all known methods of clearing the duct, this technique was applied and 3 further large stones were released from the proximal hepatic ducts. We have no experience of the cholelithophone, which amplifies the sound of a bougie touching and scraping against a stone.

If a stone remains impacted in the lower end of the common duct, it may be removed transduodenally. The duodenum and head of the pancreas must be very thoroughly mobilized, the 2nd part of the duodenum incised longitudinally, the ampulla incised, the stone removed, and the duodenotomy closed transversely in two layers. If pancreatitis is present, the main pancreatic duct may also be searched for and dilated.

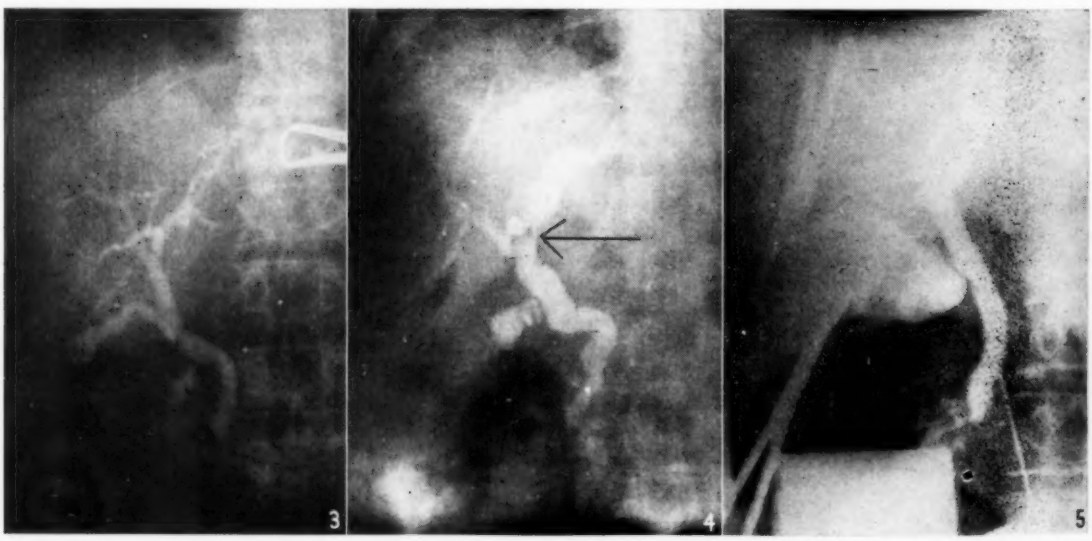

Fig. 3. Normal operative cholangiogram. *Fig.* 4. Operative cholangiogram showing stone. *Fig.* 5. Operative cholangiogram showing no stones. Common duct exploration, however, revealed a stone.

Operative Cholangiogram

This appears to be an ideal method of detecting stones in the common duct. We have given the method a thorough trial and can testify, not only to its uses, but also to its limitations.

An X-ray plate covered by a Potter-Bucky grid is placed behind the right lower rib in a special casette-holder, and the tube over the patient. When the gall-bladder and common duct have been exposed, all metal clips and instruments are removed, and 5, 10 or up to 20 c.c. of 35% Pyelosil are injected into the common duct, and an exposure is made. The operation proceeds, and the common duct is opened if any indication exists. The usual routine is followed, a T-tube is sewn tightly into the common duct, and a control cholangiogram is taken before the abdomen is closed.

We started originally with 70% Pyelosil, and injected 10 c.c. or more, but we found that stones could be missed by being surrounded by dye casting a dense shadow. We got more satisfactory pictures with a smaller amount of dye, of lower concentration.

Fig. 3 shows a normal operative cholangiogram. Fig. 4 shows stone in the common duct. Fig. 5 shows an operative cholangiogram with negative findings. In the last case the common duct was opened and a stone was found. This case indicates how misleading the information obtained in this manner may be.

Fig. 6 shows a post-operative cholangiogram taken 14 days after operation. It shows a stone in the right hepatic duct. An operative cholangiogram was not done in this case, and it is certain that it would have been of great value if it had been done before closing the abdomen. Fig. 7 shows an apparently normal cholangiogram, yet the common duct was opened and 'gravel' removed. Fig. 8 shows the dilated common duct in chronic pancreatitis.

We have had a case where a cholangiogram showed two stones in the common duct. Three stones were removed from the duct, and a cholangiogram 14 days later showed yet another residual stone!

We have many examples where the investigation was of great value but, as can be seen from the examples quoted, one may be misled in some cases. A factor also to be borne in mind is the difficulty of interpretation in some of the photographs obtained.

We have concluded that operative cholangiography is a valuable additional measure in our efforts to detect common-duct stones, and we use it as a routine. At the same time, we realize its limitations and do not use it as a substitute for other measures as outlined in this article. We have no experience of cholangio-manometry.

Generally, when a common duct has been opened, it should be drained by a soft T-tube. If a sphincterotomy has been performed, one limb of the T-tube should lie through the ampulla into the duodenum. This tube may be left in for many months, whereas the usual short-limbed T-tube is left in for an average of 14 days, when it may be removed if the cholangiogram is satisfactory or when, after clamping the vertical limb for 2 days, the patient experiences no discomfort or jaundice.

Post-operative Cholangiogram

It is possible, in spite of the most careful search at the time of the operation, to overlook one or more stones in the common duct. At the estimated time of removal of the tube, it is first clamped for increasing periods each day; if there is no discomfort, pain or jaundice, the tube may be removed, but undetected stones may still be present, and they may be responsible for post-cholecystectomy symptoms.

One may also, however, perform a cholangiogram at the estimated time of removal of the tube. The T-tube

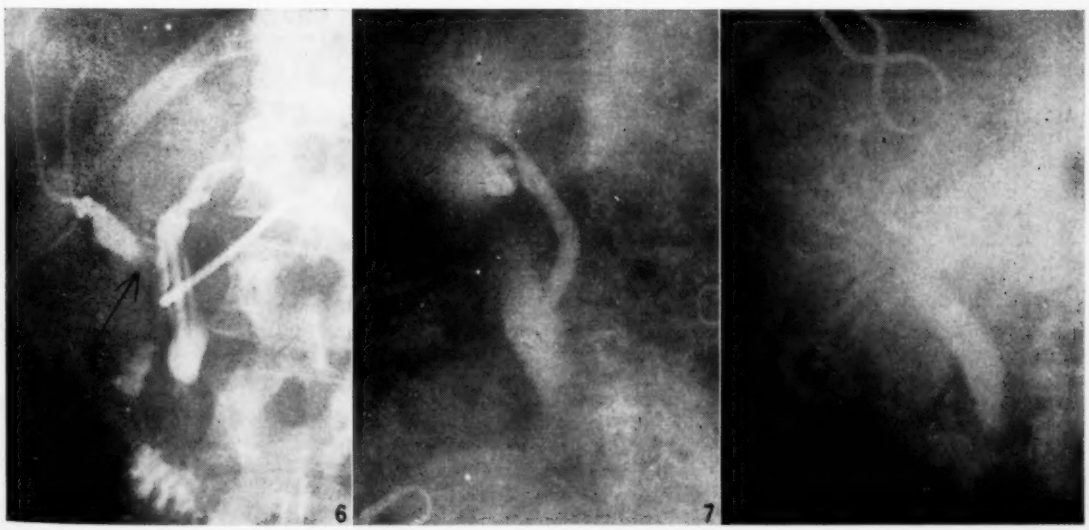

Fig. 6. 14-days post-operative cholangiogram, showing stone in right hepatic duct. Operative cholangiogram was not done in this case, but would have been of great value. *Fig.* 7. Normal operative cholangiogram, yet common duct contained gravel and debris. *Fig.* 8. Dilated common duct in chronic pancreatitis (operative cholangiogram).

is first filled with saline to eliminate air-bubbles, and a contrast medium is injected under fluoroscopic control. Exposures are made as required. If no stones are detected, if the dye enters the duodenum freely, and if there is no undue dilatation of the ducts, the T-tube may be removed.

Fig. 9 shows a normal post-operative cholangiogram.

What if stone or stones are detected in a cholangiogram taken 14 days post-operatively? Fig. 10 shows such a case. Early in our experience we used to re-operate on such a case right away; a more difficult procedure can hardly be imagined! A re-exploration at this stage may be accompanied by much morbidity, and even mortality. We think it is far safer to make a note of the presence of stones, clamp the T-tube and, if there is no discomfort or jaundice, remove it. The stones may subsequently pass, with or without pain, or if they should later cause obstructive symptoms they may then safely be removed. The stones may, of course, be removed at any time subsequently, when the patient's general condition allows, without waiting for symptoms. Biligrafin examinations may show whether stones are still present at any given time.

The results of injection of various relaxants and stone solvents through the T-tube, in an effort to relax the sphincter of Oddi and to dissolve the stone, have been disappointing in our hands. Dr. T. Marais, of our department, has tried many stone solvents *in vivo* and *in vitro*, without success.

Sphincterotomy

At times, the duodenum has to be opened in its 2nd part, the ampulla found, and the sphincter of Oddi divided. The indication for sphincterotomy to remove a stone impacted in the lower end of the duct is quite clear. Another absolute indication is when common-duct stones have been removed, or especially when recurrent stones have been removed, and it is found that the sphincter cannot be dilated more than 3 or 4 mm. (Bake size). The duct does not drain properly, and stones will surely form again unless a sphincterotomy is performed. Fig. 11 shows a case.

There is another condition, which may be termed 'fibrosis of the sphincter of Oddi', for which sphincterotomy is indicated. This may be found in patients in whom laboratory and X-ray findings are consistently negative and yet have classical attacks of biliary colic, sometimes accompanied by jaundice. This also applies to cases where, after a cholecystectomy, the patient has attacks of pain without jaundice and Biligrafin examinations are repeatedly negative. If a laparotomy is performed, other pathological conditions must be very carefully excluded, and finally the common duct should be opened. If the ampulla will not admit more than 3-mm. Bake dilator, a sphincterotomy is indicated. If chronic pancreatitis is present, the pancreatic duct should also be searched for and dilated.

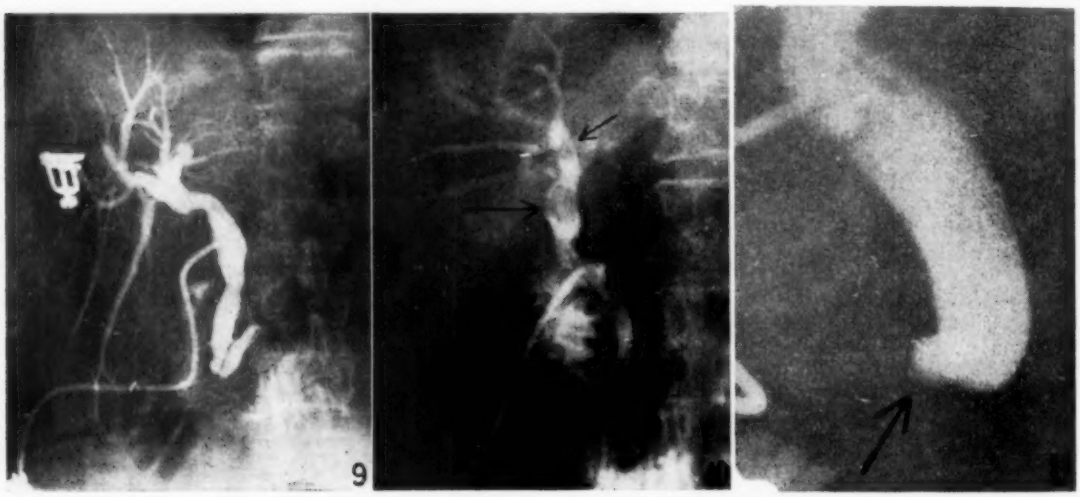

Fig. 9. Normal post-operative cholangiogram. Note filling of pancreatic duct with dye. *Fig.* 10. Post-operative cholangiogram showing residual stones. *Fig.* 11. Cholangiogram following removal of recurrent common-duct stones. Sphincterotomy should be performed. Arrow points to stenotic ampulla.

After sphincterotomy, a T-tube is inserted into the common duct, with a short limb in the duct towards the liver, and a long limb leading through the ampulla into the duodenum. This is left in for a period between 3 weeks and 3 months.

'Biliary dyskinesia' is, in our opinion, a questionable diagnosis to make. One should continue searching for an organic cause for severe attacks of pain.

SUMMARY

1. The clinical diagnosis of stone in the common duct is discussed, with special reference to its differentiation

from obstruction due to pressure from the head of the pancreas, and from infective hepatitis.

2. The operative procedure of exploration of the common bile duct for stone is discussed in detail.

3. Operative cholangiography is described and discussed.

4. Post-operative cholangiography is discussed.

5. Sphincterotomy of the sphincter of Oddi is discussed.

SAMEVATTING

1. Die kliniese diagnose van stene in die ductus choledochus word bespreek, met besondere verwysing na die differensiële diagnose van druk deur die pankreaskop, en infektiewe hepatitis.

2. Die stappe by die operasie van eksplorasie van die ductus choledochus word bespreek.

3. Operatiewe cholangiografie word bespreek.

4. Na-operatiewe cholangiografie word bespreek.

5. Deursnyding van die Oddi sphincter word bespreek.

III. BENIGN STRICTURES OF THE COMMON BILE-DUCT

This part deals with obstruction of the common bile-duct which is due to the formation of a fibrous stricture, the most frequent cause of which is operative trauma.

The history in this type of case is remarkably constant. An operation for removal of the gall-bladder is performed. There is troublesome bleeding, which is controlled with difficulty, and often frequent applications of haemostats are made and ligatures placed. Post-operatively there is leakage of bile for a variable period and, when bile drainage ceases, jaundice occurs, which may increase in severity if obstruction is complete, or may vary in intensity if, as often happens, spontaneous internal fistula occurs.

Eleven cases of benign stricture which came under my treatment are here reported in summary form, to indicate the tragic course which many of these cases run.

Repair of a stricture involves an operation which may be one of the most difficult in surgery. The most satisfactory operation is one in which the proximal and distal ends of the duct are dissected out, and an end-to-end, mucosa-to-mucosa anastomosis is made over a T-tube. If this is not possible, the proximal hepatic duct should be isolated, if necessary from within the liver substance, and anastomosed to the jejunum over a rubber or polythene tube. Frequently the patient has had a few previous attempts at repair, the liver is enlarged, and portal hypertension may be present, making the operation hazardous and one of extreme difficulty.

CASE SUMMARIES

Note: In all cases the initial one or more operations were performed elsewhere before being referred. The final operation in case 3 was performed by Dr. Lahey, and in cases 5 and 6 by Dr. Cattell, both of Boston, U.S.A. In the other cases, treatment was completed personally by the author.

Case 1. L.G.C.L., male, aged 36.

10 *April* 1944. Operation 1: Cholecystectomy.
12 *April*. Patient becoming jaundiced.
25 *April*. Blood bilirubin 10 mg.%.
27 *April*. Operation 2: Laparotomy, T-tube inserted.
May. Biliary fistula present. Patient has lost 40 lb. in weight.
July. Operation 3: Entero-enterostomy in preparation for future anastomosis.
August 1944. Operation 4: Biliary fistula implanted into duodenum.
March 1945. Operation 5: Flanged perspex tube used to bridge gap between common duct and duodenum.
September 1947. Operation 6: Common hepatic duct anastomosed to previously-prepared jejunal loop.
July 1948. Haematemesis. Portal hypertension present.
December 1948. Operation 7: Jejunal loop converted into Roux-Y type.
Patient now a morphine addict.
8 *July* 1951. Died. Massive haematemesis from oesophageal varices.

Case 2. Mrs. L.H.W., aged 66.

November 1948. Operation 1: Cholecystectomy and removal of stones from common duct.
June 1949. Pain and obstructive jaundice.
July. Operation 2: Common-duct stricture 2 cm. above papilla. Stricture incised, long-limb T-tube inserted.
October 1949. T-tube removed.
February 1950. Recurrence of pain, rigors and jaundice.
June 1950. Operation 3: Common-duct stricture. Anastomosis performed between common duct and duodenum.
Follow-up: Has remained completely well; last examined in June 1955.

Case 3. Mrs. L., aged 45.

October 1947. Operation 1: Cholecystectomy.
October (5 days later). Operation 2: Wound disruption sutured. Bile draining.
November 1947. Operation 3: Common duct sutured over T-tube.
January 1948. Operation 4: Anastomosis duodenum to common bile-duct.
February 1949. Operation 5: Roux-Y hepatico-jejunostomy.
September 1950. Operation 6: Vitallium tube inserted in anastomosis.
May 1952. Operation 7: Exploration: Inoperable: portal hypertension; external biliary fistula established.
July 1952. Died. Portal hypertension.

Case 4. Mrs. A.S.S., aged 56.

August 1951. Operation 1: Cholecystectomy, followed by biliary fistula and jaundice.
September. Operation 2: Laparotomy and drainage to establish biliary fistula.
November 1951. Operation 3: Attempted anastomosis of hepatic duct to stomach.
May 1952. Operation 4: Excision of stricture and end-to-end suture of duct over T-tube. Liver cirrhosis and portal hypertension present.
February 1953. Operation 5: Removal and re-insertion of new T-tube because of blockage. Vitallium tube attached to upper limb of T-tube.
January 1954. Operation 6: Laparotomy for removal of tubes. Liver cirrhotic, function poor.
11 *days later*. Died in hepatic coma.

Case 5. Mrs. I.C.N., aged 27.

October 1952. Operation 1: Cholecystectomy, followed by bile leakage.
November 1952. Operation 2: Drainage of bile accumulation.
April 1953. Operation 3: Attempted common-duct repair.
October 1953. Operation 4: Exploration. Common duct exposed. Uncontrollable bleeding due to portal hypertension.

November. Operation 5: Gastrectomy and splenectomy for uncontrollable haematemesis.
December 1953. Operation 6: Anastomosis common hepatic duct to duodenum.
Follow-up: June 1955. Has gained 34 lb. in weight, feels well. Slight recurrent jaundice (anastomosis probably narrowed).

Case 6. Mrs. H.C.V., aged 51.

September 1952. Operation 1: Cholecystectomy, followed by bile drainage and jaundice.
November 1952. Operation 2: Common duct repair.
April 1953. Operation 3: Attempted common duct repair.
Attacks of rigors and jaundice continued.
June 1954. Operation 4: Anastomosis left hepatic duct to jejunum, with entero-anastomosis.
Follow-up: September 1955. Very well; has gained 27 lb in weight.

Case 7. Mrs. S.E.P., aged 54.

March 1954. Operation 1: Appendectomy, cholecystectomy, common duct cut, recognized, sutured over T-tube, vertical limb coming out through the suture line.
6 *weeks later*. T-tube removed.
October. Obstructive jaundice developed.
November 1954. Operation 2: Excision of stricture, end-to-end suture of common duct. T-tube brought out through a separate stab above the anastomosis.
August 1955. T-tube removed.
October 1955. Patient remains well.

Case 8. Mrs. A.J.L. v.d. W., aged 54.

April 1954. Operation 1: Cholecystectomy. Troublesome bleeding.
June. Intermittent jaundice and fever.
August. Continuous pain right upper quadrant, jaundice, itching, vomiting.
September 1954. Operation 2: Stricture of common hepatic duct high in liver hilum. Difficult anastomosis of hepatic duct to a loop of jejunum.
5th post-operative day. Died, ? bile peritonitis.

Case 9. Mrs. S., aged 47.

12 *March* 1955. Operation 1: Cholecystectomy. Great technical difficulty; bleeding; massive ligature applications. Post-operative bile-leakage.
4 *April*. Operation 2: Drainage bile peritonitis.
12 *April*. Operation 3: Drainage bile accumulation.
3 *May*. Operation 4: Anastomosis left hepatic duct to loop of jejunum, with entero-anastomosis.
October 1955. *Follow-up:* Completely well and free of jaundice.

Case 10. Mrs. L.M.S., aged 36.

19 *August* 1955. Operation 1: Cholecystectomy.
20 *August*. Jaundice; no bile leakage.
23 *August* 1955. Operation 2: Common duct found divided and tied off. End-to-end anastomosis over T-tube, after removing 10 stones from distal common duct.
Follow-up: Doing well. T-tube remains in position.

Case 11. Mrs. A.M.M., aged 57.

10 *February* 1955. Operation 1: Cholecystectomy.
30 *March*. Operation 2: Drainage posterior collection.
7 *July*. Operation 3: Drainage anterior collection.
September. Polythene tube passed, jaundice.
15 *September* 1955. Operation 4: End-to-end anastomosis common hepatic duct over T-tube for high stricture at bifurcation of hepatic duct.
Patient remains well.

It is hoped that a mere descriptive summary of these cases will suffice to indicate what a real tragedy it is to sustain an injury to the common bile-duct, and at the same time what tremendous problems arise in surgical therapy. Probably the only unavoidable strictures are those which occur as the result of ulceration of the duct following long-standing impaction of stones or pressure from without, as by aneurysm. Such cases undoubtedly occur; case 2 is an example. It is also stated that bile leakage around the common duct may lead to sclerosis.

Trauma to the common duct during routine cholecystectomy is avoidable, and the following points are considered to be important in this respect:

1. Surgery of the gall-bladder should not be undertaken by any person who has not been trained in biliary surgery.
2. Good and safe anaesthesia, with adequate relaxation.
3. Good exposure through adequate incision.
4. Ample and good assistance at operation.
5. Good light. Movable and adjustable spotlights are more satisfactory than a single overhead lamp.
6. Careful exposure of all anatomical structures in the region of the neck of the gall-bladder before division of the cystic artery and cystic duct. If there is any difficulty at all in identifying the structures, removal of the gall-bladder should start at the fundus, and proceed down towards the cystic duct. One should bear in mind congenital abnormalities of the ducts, such as accessory hepatic ducts, and anomalous position of the arteries, especially the right hepatic artery. If there is still difficulty in identifying structures, either a cholecystostomy should be performed, or the gall-bladder should be slit open, most of it removed, and the remaining mucosa cauterized.
7. If a sudden severe bleeding should occur, wild clamping with artery forceps should be avoided; the bleeding should be controlled with packing, pressure, and patience, until it is possible to pick up no more than the bleeding point itself, thus avoiding injury to the bile ducts.

PRINCIPLES OF REPAIR OF COMMON BILE-DUCT STRICTURES

Thorough pre-operative evaluation of the general condition of the patient should be made, with special attention to liver and kidney function. Thorough pre-operative preparation should be along standard lines, with a high-protein, high-carbohydrate, and high-vitamin administration, by mouth as well as parenterally. Jaundiced cases should have routine antibiotic administration, as well as blood, and vitamin K1, intravenously.

Complete exposure of the area is essential; this may be the most trying part of the operation. A long right paramedian incision is made, the peritoneal cavity entered low, and adhesions between the scars of the previous operation and the viscera separated from below upwards.

The next step is to separate the hepatic flexure from the under surface of the liver and lateral abdominal wall and turn it medially and downwards. The transverse mesocolon is separated from the duodenum and head of the pancreas at the same time. This procedure exposes the 2nd and 3rd parts of the duodenum, which are now completely mobilized, together with the head of the pancreas. Next the stomach and pylorus are separated from the under surface of the liver and 'rolled' downwards, together with the first portion of the duodenum. This exposes the gastro-hepatic omentum.

The structures in the gastro-hepatic omentum are now identified, and this part of the operation may again be very trying. It is often accompanied by much troublesome bleeding. The distal end of the common bile-duct is sought first, dissected free for about 2 cm., and the patency of duct and ampulla and freedom from stones is demonstrated by bougies, and may be confirmed by cholangiography. Next the stump of the hepatic duct is sought in the hilum of the liver. It may be necessary to core out the hilum with the diathermy loop to obtain an adequate stump of hepatic duct. The duct is opened, dilated, and flushed out, and cholangiogram may again be useful.

If at all possible, a primary anastomosis is performed between the two ends of the duct, over a T-tube the long limb of which emerges from the duct at a point above or below the anastomosis. The T-tube is left in for a year, giving the fibrous ring at the anastomosis time to become moulded around it. The tube is washed out daily with saline solution, and a cholagogue is given by mouth.

Unfortunately, the ideal as outlined above is frequently not possible. The most satisfactory alternative is then to perform an anastomosis between the stump of the hepatic duct and a loop of jejunum, which can be brought up ante-colically without tension. A jejuno-jejunostomy is also performed. Other less satisfactory alternatives have been described.

Even with adequate operations, the anastomoses are inclined to shrink, necessitating re-operation in some cases. Some of these cases develop biliary cirrhosis with portal hypertension, and eventually succumb.

Traumatic stricture of the common bile-duct is a serious but preventable condition, and it behoves every person who performs a cholecystectomy to exercise the greatest possible care to prevent damage to the bile passages.

SUMMARY

1. Benign stricture of the common duct follows most frequently on trauma sustained during operation of cholecystectomy.

2. Eleven cases of benign stricture of the common bile duct are reported in summary form.

3. Essential points in prevention of trauma to the bile ducts during cholecystectomy are stressed.

4. Principles of secondary repair of common-duct strictures are outlined.

SAMEVATTING

1. Benigne strikture van die ductus choledochus volg meestal op besering aangewend gedurende die operasie van verwydering van die galblaas.

2. Elf gevalle van ductus choledochus-striktuur word in hierdie artikel gerapporteer.

3. Belangrike punte in die verhoeding van besering aan die galbuise gedurende galblaas-operasies word genoem en beklemtoon.

4. Prinsiepes van sekondêre hegting van galbuis-striktuur word aangegee.

ENDOCRINOLOGICAL PROBLEMS : WHAT CAN BE LEARNED AT THE BEDSIDE*

I. FREED, M.B., B.Ch. (RAND)

Johannesburg

With the many new technological advances and therapeutic weapons which have become available in various fields of medicine in recent years, the responsibility of the doctor towards his patient becomes greater than it has ever been before. Only by the continual process of self-education can the physician engaged in the practice of medicine best fulfil his role in caring for the sick. By remaining abreast of new information with regard to the basic sciences and by integrating them with his observations and examinations at the bedside, it is possible to bring to the patient the maximum amount of in elligent medical assistance.

The field of endocrinology is only one of many in which such principles apply and it is the purpose of this paper to show how some of the problems may present themselves to a clinician at the bedside without recourse in the first instance to the various laboratory or other investigative procedures which may be necessary in their more exact elucidation. In the limited time available it will not be possible to deal with more than a very few of the various clinical pictures which may present themselves.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

HYPERTHYROIDISM

The thyrotoxic individual, usually a female, is nervous or irritable, restless and fidgety. Commonly when one approaches the bedside, she jumps into an upright position and throws off the bedclothes. She may wish to cooperate but rather overdoes it. Such behaviour constitutes a pattern which is almost pathognomonic. The patient feels hot and likes to be in a cold environment. Handshaking gives a nearly diagnostic impression; the hand is very hot and moist—sometimes actually dripping wet. Whilst occasionally such hands may be found in other conditions, the converse—the finding of a cold hand, dry or moist—almost excludes hyperfunction of the thyroid. Redness of the elbows, first noted by Plummer, is very frequently present and also of the knuckles and thenar and hypothenar eminences. The fingernails tend to leave the nail-bed, giving a concave or wavy margin to the terminal portion in contact with the nail-bed. Pigmentation of a more or

less Addisonian variety is met with occasionally, but the mucous membranes remain free. Then there are, of course, the eye signs, which may be related either (1) to lid retraction, causing the wide palpebral aperture or the lid lag or the staring or frightened expression, or (2) to extrinsic muscle weakness, causing limitation of movement of the eyeball, especially in a upward direction, or diplopia, or (3) to swelling—chemosis of the conjunctiva or swelling of the lids. It is important to know that these eye signs vary independently of the intensity of the thyrotoxicosis and that in certain cases they may get worse when the thyrotoxicosis is subsiding. Burning, photophobia, epiphora, corneal ulceration and marked blepharitis may occur. Often exophthalmos is obvious with the patient upright but is almost unnoticeable when the patient lies on his back. The tongue, apart from exhibiting a definite tremor, is often red and smooth owing to achlorhydria.

The neck, of course, often shows the goitre, but it is possible for marked thyrotoxicosis to exist without visible or palpable goitre. The carotid arteries are often to be seen violently throbbing. Various muscular phenomena are frequently observed. A useful test for the demonstration of muscular weakness was described by Lahey in 1926: If a thyrotoxic patient seated in a chair is asked to hold one leg out straight in a horizontal position, he can do so for 25-30 seconds only, whereas most normals can go over 60 seconds. Myasthenia gravis occurs in some, and other degrees of weakness in 75% of all cases of thyrotoxicosis. Acute bulbar palsy is sometimes seen. In a considerable number of cases wasting of the temporalis muscles and the interossei of the hands is seen; in a few cases, wasting of all skeletal muscles. Vitiligo, alopecia and decrease of the auxillary hair, and premature graying of the scalp hair are other features which are sometimes observed. Not infrequently neuropsychiatric manifestations may be the presenting features—paraphasia, acalculia, psychosis with hallucinations, bulbar palsy, coma, myo-encephalopathy, choreo-athetotic movements—and often these indicate the presence of the so-called 'thyroid crisis'. Then there are the easily recognized disturbances referable to the cardio-vascular system which may be elicited on examination—the tachycardia which is present during waking and sleeping hours, premature beats or paroxysms of auricular fibrillation, a forceful cardiac apical impulse, a systolic murmur heard all over the praecordium, a raised systolic blood-pressure and a slightly decreased diastolic pressure with a resultant high pulse-pressure.

Finally, I wish to mention the condition of pretibial myxoedema, which occurs almost invariably in patients with severe or malignant exophthalmos and often after subtotal thyroidectomy. It appears in painless symmetrical patches of variable size but of definite outline on the anterior aspects of the lower half of the legs or the dorsal surfaces of the feet. It has also been described on the face, the eyelids and the scrotum. The skin overlying the lesions is thickened and pigskin-like in appearance, and red cyanotic or yellow-white in colour. It does not pit on pressure but will dimple after the local injection of hyaluronidase, and its cause is probably related to an excessive production of thyrotropic hormone.

HYPOTHYROIDISM

Myxoedema. In the adult case of hypothyroidism also, interestingly enough, the patient is generally a female. Myxoedema is, in fact, 4 times as common in females as in males. It is well to remember that the disease is extremely insidious in its onset and many years of thyroid insufficiency or deficiency may be required before myxoedema becomes manifest. It would be pertinent to mention at this stage that if the thyroid gland of a person with standard metabolism is extirpated, it will take 20 days for the metabolic rate to decrease to approximately minus 20, when mild clinical manifestations of hypothyroidism begin to appear. After 40 days the rate will be about minus 30 and frank myxoedema begins to appear. Within 80 days the disease is full blown. Decreased sweating and increased sensitivity to cold are the early manifestations. The patient wants more clothes, more cover and more heat in his room. Drowsiness and slowing of mental and physical activity appear, soon followed by lack of energy and ambition, and puffiness of the skin. Peri-orbital puffiness is particularly striking. Mucus collects in the eyes and the lids often are found on waking to be stuck together. The skin becomes pallid and may have a yellow hue. The soft tissues of the body become thickened, particularly noticeable in the face and hands. The nose, ears, lips and other portions of the face appear large. The face looks somewhat expressionless. The tongue is large and the voice husky, leathery and low-pitched. The speech is slow, deliberate, without inflection. Some words sound slurred or otherwise inarticulate. The patient may lose some of the body hair and develop constipation, meteorism, deafness, oedema, dyspnoea, cardiac enlargement. Menorrhagia occurs very frequently. The pulse rate is slow. The blood pressure is generally raised.

Cretinism. Hypothyroidism which develops before birth and produces cretinism either of the endemic or the sporadic type is an entity which can often be recognized at the bedside in the latter half of the 1st year of life and sometimes as early as the 3rd month. The segment of the body above the symphysis is distinctly longer than that below. The head is large in comparison to the body and closure of the fontanelles is considerably delayed. The forehead is short, the nose broad, the eyes wide apart, the eyelids puffy and wrinkled, the mouth open, the lips and tongue thick, and the voice deep and husky. The teeth appear late, the neck is short and thick, and the hands and feet are puffy, short and broad. The abdomen is large and protuberant; an umbilical hernia is often present. The skin is dry and cool, pale and thick, and roughened. The hair is sparse, coarse and dry. Sitting, walking and talking are considerably delayed. Most important of all, when one wishes to confirm one's diagnostic impression by means other than bedside observations, it must be remembered that the appearance of the ossification centres is markedly delayed.

Juvenile myxoedema. When hypothyroidism develops after birth, the condition of juvenile myxoedema results,

and its characteristics are midway between those of cretinism and adult myxoedema; growth and development, though normal in the early stages of life are retarded, and dwarfism and delayed sexual development become apparent. The extremity-trunk ratio is infantilistic, i.e. the ratio of the distance from the symphysis to the head to that from the symphysis to the feet remains 1·7/1 as it is at birth. This is a simple yet valuable bedside observation to make which, when elicited, will readily lead one to perform all the other investigations —biochemical and radiological—for the definitive diagnosis of this condition.

HYPERFUNCTION OF THE SUPRARENAL CORTEX

Before describing the features in any detail, it would be as well to clarify the terms and nomenclature used at the present time:

Cushing's syndrome refers to the clinical picture designated by Cushing as pituitary basophilism; it is encountered chiefly among young women, although it does of course occur in men and in children of either sex. Its salient features are a distinctive habitus characterized by wasting of muscle and obesity or an abnormal distribution of fat, the combination of which makes the trunk, face and neck appear obese and the extremities thin; hirsutism; diabetes either latent or frank; ecchymoses; and amenorrhoea or impotence. Except for the presence of hirsutism, there is little or no evidence of masculinization. Usually there is a pad of fat over the cervico-thoracic part of the spinal column. The upper lids and corners of the mouth droop. The cheeks may be fiery red and so full that they obscure the ears when the face is viewed squarely from the front. In children, the normally short neck may be completely obscured by a drooping double chin. As the fat cheeks roll forward and hang down, the mouth appears unduly small and set deep in the face. Almost always there is an easily demonstrable muscular weakness, sometimes of a degree seen in moderately advanced muscular dystrophy or atrophy. Acne of varying intensity is fairly common. Generally the skin is thin, mottled and high coloured, and there are distinctive purplish striations in it.

Adrenal virilism and androgenital syndrome are more or less synonymous terms denoting a clinical picture which usually occurs among young women, but again it may occur at any time of life and in either sex. It is characterised by most of the physical attributes of masculinity, namely vigour, muscularity, enlargement of the larynx and hirsutism of the type normally occurring in men. When this syndrome occurs prenatally in a foetus having ovaries, it is associated with anatomic defects of the genital system, and the clinical picture is then known as feminine pseudo-hermaphroditism. When it occurs postnatally among children, the syndrome is characterized usually by somatic and sexual precocity. Among boys this sexual precocity is homologous, i.e. masculine in type; in the case of girls, the puberty is heterologous, which means that it proceeds along masculine lines. Among children of either sex a distinctive habitus appears as the child grows older. The arms and legs are short and the trunk is relatively elongated. The muscles bulge conspicuously, the shoulders are broad and the hips are narrow. When the syndrome occurs in a woman she becomes more or less masculinized. Menses cease and the breasts become smaller.

To amplify some of the features of the *Cushing's syndrome* from the point of view of their importance at the bedside: The hypertension which occurs may be relatively benign. On the other hand it may be so severe that it may be associated with heart failure or cerebrovascular accident, or blurring of vision or blindness. The obesity will be very extreme when the disease occurs in childhood. The diabetes which sometimes occurs may be an insulin-resistant type. Osteoporosis may manifest itself to the clinician as severe backache with root pains associated with compression fracture. Attacks of renal colic may draw attention to the hypercalciuria which is the sequela of the osteoporosis. Muscular weakness occurs in the Cushing's type and is particularly noticeable when the patient tries to climb stairs. It should be remembered, however, that the actual atrophy of the muscle may be hidden by the overlying fat. In the adreno-genital type there is increased strength and vigour. The purple striae of the skin (see above) occur in the lower part of the abdomen, the anterior lateral aspect of the thighs, the upper part of the arms, the axillae, the pectoral regions, and even the breasts. They are roughly parallel and depressed and vary in size from 0·5 to 5 cm. in width and 5 to 15 cm. in length. They may become necrotic and infected. In addition to the striae, the entire skin of the body often becomes thin and transparent, so that the superficial veins are unduly prominent. Keratosis pilaris in the region of the buttocks makes the skin of this area feel like sandpaper. Ecchymoses and petechiae appear after the slightest trauma and are often prominent symptoms. The face may be fiery red and the skin of the extremities may be mottled and somewhat cyanotic (see above). Beneath the clavicles and over the sternum there is often a semilunar area containing tiny whitish papules, between which the skin is red and flushed. This has been referred to as the 'androgenic flush'.

In connection with the *adreno-genital syndrome*, I should like to make a few points which may be of assistance to the clinician at the bedside in helping him to formulate an assessment of the picture: Simple hirsutism in the absence of virilism rarely proves to be adrenal cortical in origin. Patients with hirsutism plus obesity and amenorrhoea deserve further diagnostic study, even though few of them prove to have recognizable organic adrenal cortical disease. Patients with both hirsutism and milder degrees of virilism are more likely to have organic adrenal cortical disease. A feminine pseudo-hermaphrodite almost always has adrenal cortical hyperplasia rather than tumour. Most patients who show all the features of Cushing's syndrome and nothing else prove to have adrenal cortical hyperplasia rather than adrenal cortical tumour. Patients who present mixed clinical pictures of both the adreno-genital syndrome and Cushing's syndrome are likely to have adrenal cortical tumours rather than hyperplastic cortical lesions. An enlarged clitoris in a patient who seems to have nothing but Cushing's syndrome indicates adrenal cortical tumour; however, an enlarged clitoris

in a patient who has the adreno-genital syndrome may indicate either tumour or hyperplasia.

ADDISON'S DISEASE

Turning from hyperfunction of the suprarenal cortex to hypofunction, or Addison's disease, the clinical description given by Addison himself exactly one hundred years ago is still the best: 'An insidious onset of a feeling of languor and debility, a remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change in the colour of the skin.'

The classical symptoms and signs are the profound asthenia, the gastro-intestinal symptoms, particularly anorexia, nausea, vomiting, constipation or diarrhoea and occasionally relatively severe abdominal pains, marked weight loss, pigmentation, and hypotension. There is a slow but continued loss of weight and the patient either becomes aware himself or is told by others of a change in the colour of his skin. Curious mental changes characterized by marked irritability and personality alterations often severe enough to suggest an underlying psychosis of a persecutory character frequently appear. Occasionally the first presenting evidence of the disease is the development of the so-called Addisonian crisis, precipitated by an acute infection or some surgical procedure. It is well to remember that pigmentation and hypotension may be present for months or years—even 15 years—before gastro-intestinal symptoms and weight loss appear. The pigmentation, which is invariably present but not always so, often begins as a sun-tan which persists and becomes permanent and with the passage of time becomes progressively darker.

The occurrence of pigmentation in unexposed portions of the body is of great clinical significance. First in importance and frequency are the unshapely patches of brown-grey, grey-black or blue-grey pigmentation observed on the mucous membrane of the oral cavity, particularly on the lips, gums and tongue, and occasionally on the posterior pharyngeal wall. It is to be borne in mind that oral pigmentation occurs in argyria and that it is a normal occurrence in the Bantu. The areolar, peri-anal and genital pigmentation shows an increase in its intensity; also, operation scars, pressure points such as the elbows, the hat-band region, and places where a restraining or tight-fitting garment has caused pressure, will show the appearance of this pigmentation. The folds of the axillae, the palms of the hands, and the knuckles of the fingers are other frequent sites. In addition to the diffuse pigmentation, many patients develop scattered jet-black spots or freckles. Infrequently mahogany-coloured longitudinal bands of pigmentation appear in the finger nails. Addison himself also called attention to areas of vitiligo or leukoderma which were observed as small decoloured patches on the back and trunk.

The hypotension frequently has a postural component and the symptoms associated with it thus constitute a considerable part of the symtomalotogy. The dizziness, blurring of vision, sense of faintness, cardiac palpitation and tachycardia, and occasionally even angina, occur particularly with change of posture and most frequently on rising in the morning. The loss of weight may be considerable and yet the patient does not appear to be Cachectic. This was also commented on by Addison in his original description. *The asthenia* is profound. The patient both looks and acts unutterably wearied even when lying in bed. He may be completely incapable of any effort and no amount of rest in itself produces any improvement in this symptom. The crisis is heralded by an intensification of all the previously mentioned symptoms and signs. The weakness is more profound, the blood pressure falls, the skin colour assumes a darker hue with a superimposed dusky slaty cyanosis. Nausea, vomiting and diarrhoea becomes severe, and sometimes intractable abdominal pain may develop. A thready, feeble pulse, distant and muffled heart-sounds, a markedly dehydrated skin, soft and sunken eyeballs, all indicate the state of circulatory collapse which has developed.

PHAEOCHROMOCYTOMA

A clinical syndrome concerning the chromaffin tissue of the medullary portion of the suprarenal gland may be mentioned here, viz. that produced by the pheochromocytoma. If recognized, it presents one of the remediable causes of hypertension. It manifests itself frequently in the form of attacks of *palpitation*, headache, dizziness, blurring of vision, epigastric and thoracic pain, pain in the extremities and back, dyspnoea, choking sensations, nausea, vomiting, diarrhoea, profuse sweating, shakiness, and fear. The face and extremities may be pale or cyanotic. The pulse is small and hard. The blood pressure during the attack may be more than 300 mm. mercury. The attack may last minutes or days. Change of posture, mechanical pressure on one or other kidney area, hyperventilation, pain, emotion, micturition, may bring on an attack. Between attacks, the patient enjoys a sense of ordinary health. Sometimes, the elevation of blood pressure is persistent.

UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 9 February 1956.

Plague, Smallpox: Nil.

Typhus Fever, Cape Province: One (1) European case in the Queenstown Municipal area. Diagnosis confirmed by laboratory tests. The case of typhus fever notified in bulletin No. 4 of 1956, occurred in the Umtata district and not the Qumbu district as stated. *Transvaal:* One (1) Native case in the Rustenburg district. Diagnosis confirmed by laboratory tests.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Dacca (Pakistan).

Smallpox in Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Allahabad, Bombay, Delhi, Kanpur, Madras, Magpur (India); Dacca (Pakistan); Huế (Viêt-Nam).

Typhus Fever: Nil.

CYCLOPROPANE : A VINDICATION

H. H. SAMSON, *M.B.E.*, M.R.C.S. (LOND.), L.R.C.P. (ENG.)

Johannesburg

After administering approximately 10,000 cyclopropane anaesthetics, with and without pentothal and the muscle relaxants, to patients of all ages and for a great variety of operations, and including many already suffering from grave cardiac lesions, I am firmly convinced that cardiac arrhythmia occurring during the administration of this potent agent is not due, as universally believed, to the agent *per se*. I was fortunate not to have lost a single patient, although innumerable patients manifested varying degrees of collapse ('cyclo shock'), because of my initial inexperience of the closed circuit technique.

Time and again one has proved in practice that such arrhythmia is due to the ill-effects of hypoventilation—hypoxia and hypercapnia—resulting from defective technique and lack of appreciation of the condition. It has been repeatedly shown that the arrhythmia can be abolished with ease and safety by correcting these two factors, as will be described below. Of these two extraneous factors hypoxia is by far the more dangerous, and it is quite possible that those with low cardiac reserves may succumb from this cause long before the toxic effects of hypercapnia become grossly manifest. A slight rise in the blood pressure and a rather rapid pulse rate (the effects of hypoxia) are usually the early signs that all is not well. When the pulse becomes full and bounding, with a tendency to slowness, hypercapnia has joined forces with hypoxia.

Hypoventilation can produce practically every variety of abnormal cardiac irregularity. I have never encountered ventricular fibrillation. This complication, according to the pundits, is apparently bound up with chloroform, but I am strongly suspicious that it occurs only in the presence of too little oxygen.

Cyclopropane is a narcotic and *ipso facto* a central respiratory depressant—and a powerful one—but it is *not* a cardiac poison. Any poisonous effect on the heart is due to concomitant hypoventilation. For those who like formulae, the toxic set-up can be crudely expressed thus: $C_3H_6—O_2+(CO_2)n$

That is to say, the narcotic, in the presence of hypoxia and hypercapnia, can be shown to affect the heart. But the factor C_3H_6 (cyclopropane) can be removed from the closed-circuit situation and any other known anaesthetic agent substituted, excluding the halogen derivatives (because of the danger of phosgene formation), without affecting the issue. In other words, hypoxia combined with hypercapnia is liable to produce cardiac arrhythmia, *whatever the anaesthetic agent. In fact, I believe that hypoventilation* per se *can do so in the absence of narcosis*, but it is more prone to occur and with greater severity in the temporary pathological state of anaesthesia, in which the respiratory centre is always depressed, especially with powerful agents such as cyclopropane. The complicated method of administration of this agent is another contributing factor.

It should now be clear why adrenaline so dangerously aggravates the cardiac effects of hypoventilation. Its harmful effect has been ascribed (on the basis of a single death finding in dogs!) to its combination with cyclopropane,[1] but this is merely because hypoventilation is more commonly associated with the closed-circuit technique, which to many administrators implies cyclopropane. If cyclopropane is given with due regard to the prevention of hypoxia and hypercapnia, there is no reason why adrenaline may not be safely used. Indeed, it is the duty of the administrator to reassure the operator who wishes to use this useful vascular constrictor, that he may well do so without detriment to the patient. Obviously adrenaline should not be administered to any patient, anaesthetised or not, who is hypoventilating.

As regards the treatment of cardiac arrhythmia, all that is necessary is to empty the re-breathing bag of its accumulated CO_2 and to refill it repeatedly with liberal supplies of oxygen, while maintaining an increased tidal volume to ensure more rapid restoration of the normal blood levels of the respiratory gases. This procedure is carried out until the pulse returns to its pre-hypoventilating state. During the process of 'washing out' of excess CO_2 and 'filling up' with oxygen the cyclopropane can be turned off, as in practice the hypoxia and hypercapnia have themselves anaesthetized the patient sufficiently and there should be no fear of his waking up. Besides, cyclopropane is expensive and the gas will have practically no narcotic effect when so greatly diluted; also there is always the possibility of an explosion when this gas is forced out of the re-breathing bag.

If the anaesthetist is not inept and learns to prevent the onset of hypoventilation, there is no reason why cyclopropane should not be administered for hours on end, with benefit to patient and surgeon.

CONCLUSION

It appears that the extent of ventilation is too often over-estimated with the result that weird phenomena make their unwanted intrusion. It is high time, therefore, that there was a change in the teaching of anaesthesia in this respect. The normal tidal volume must be maintained with adequate oxygen intake, and excess CO_2 must be prevented at all times. This is a cardinal rule applicable not only to general anaesthesia but also to the treatment of and prevention of pulmonary conditions.

Most of the morbidity and mortality associated with narcosis can be prevented. Let us call a halt to making the *narcotic* the scapegoat of general anaesthesia.

1. Meak, W. J. (1940): Proc. Staff Meet., Mayo Clic., **15**, 237-240.

THE NATIONAL COUNCIL FOR THE CARE OF CRIPPLES IN SOUTH AFRICA—CEREBRAL PALSY DIVISION

The National Council for the Care of Cripples in South Africa (P.O. Box 10173, Johannesburg, Telephone 23-5496) has recently formed a Cerebral Palsy Division.

Nearly all of the associations and institutions working among the cerebral palsied in the Union and South West Africa, including those at Pretoria, Cape Town, East London, Port Elizabeth, Durban and Windhoek, as well as the Forest Town School for spastics, Johannesburg, are affiliated to the National Council. The object of the Cerebral Palsy Division is to co-ordinate the work on a nation-wide basis, and to promote further services for these handicapped people, of whom there are many thousands throughout the country.

The following programme of action has been adopted by the Division:

1. Recognising the urgent and imperative need for full co-operation between those bodies in South Africa working on behalf of the cerebral palsied, and the importance of complete co-ordination of programmes to prevent unnecessary duplication and waste of resources, the Division pledges itself to work for unity in this field. It also undertakes to seek the cooperation of the Government in its plans and operations.

2. Facilities for the diagnosis and treatment of cerebral palsy will be made available in the main centres of the Union and South West Africa, and all possible help will be offered to local communities in the establishment of schools, clinics, treatment centres, parents' groups and cerebral palsy associations. Immediate aid is being given to Port Elizabeth, and attention to the needs of Windhoek and East London will follow. As a guide to local groups a pamphlet will be prepared containing suggestions on how to start local projects for the cerebral palsied.

3. The education of the public as to what cerebral palsy is and how it can be effectively dealt with, and the securing of the interest and support of medical, educational and other agencies, will be initiated. As the first step in this direction it is hoped shortly to publish a journal devoted to the interests of the cerebral palsied, whilst the establishment of a library will be proceeded with as soon as possible.

4. The care and treatment of those regarded as ineducable will be carefully considered, and where possible, proceeded with, and in this connection attempts will be made to establish a recognized standard of educability.

5. The training of personnel—doctors, therapists, teachers etc.—is regarded as of vital importance if facilities for treatment and education are to be extended, and every effort will be made in cooperation with other bodies to provide such training. The appointment of orthopaedic technicians and visiting teachers will also be given careful consideration.

6. In order to assess the extent of the problem, and as a working guide in the extension of facilities for treatment, the compilation of a National Register of all cerebral palsied persons in South Africa, already begun, will be proceeded with, and to this end cooperation with other organizations will be both sought and given.

7. Attention will be given to the needs of non-Europeans afflicted with cerebral palsy, a field hitherto almost untouched, and facilities for their treatment and habilitation established as soon as possible.

8. Clinical and educational research will be carried out into the causes and methods of treatment of cerebral palsy, and the vital question of prevention as well as the necessity for early diagnosis will be given careful study.

9. The establishment of hostels in large centres, in order that children living in country areas might be given the opportunity for treatment and education, will be regarded as an essential part of the programme of the Cerebral Palsy Division.

10. Every endeavour will be made to secure employment or sheltered employment for such persons as are able to take up some form of work, and consideration of the possibility of founding a permanent Home for the very severely handicapped will be given. Full support will be given to the establishment of a Holiday Home in Natal.

IN MEMORIAM

DR. ARTHUR FELIX

Prof. A. Pijper (Pretoria) writes:

Dr. Arthur Felix, whose sudden death took place on 14 January 1956, so very typically on his way to his laboratory at the Lister Institute in the evening, should be commemorated in all medical journals. Dr. Felix was not a medical man; he had a doctor's degree in chemistry, and surely it is given to very few people not medically trained to make such a lasting imprint on medicine as he did. It is not my intention to attempt anything like a biography; that would fill pages.

His career started in a railway carriage converted into a laboratory in which he and Prof. Weil followed the Austrian troops into Russia and Turkey during the first world war. In that railway carriage the Weil-Felix reaction saw the light of day; there the conceptions of H and O, now household words in every laboratory, were worked out, and the new era in agglutination of bacteria started. Weil and Felix, notwithstanding the attacks of typhus they went through themselves, brought their railway carriage back to Vienna when their side had lost the war and communists reigned in Austria; and it was again no mean effort in the existing chaos to save all those wonderful bacterial cultures they brought back with them, of which many have survived to the present day under their well-known names (Ty 901, Ty 2, Proteus X19, Proteus X2, etc.).

There followed a perhaps quieter but not much easier period of laboratory work in the new state of Israel and then a very long stay in London at the Lister Institute. Here the new discoveries in typhus and typhoid (to put it simply) were worked out further and consolidated, and Felix became the central bureau of all work on the globe in these subjects. He guided, advised and directed numbers of people in all countries. I have often wondered how without his inspiration, example and direct guidance a way would ever have been found out of the chaos in South Africa of louse typhus, flea typhus and tick-bite fever. He was also responsible for the abolition in South Africa of the complement-fixation test for typhoid and its replacement by the O and H receptor analysis, in which, as in so many things, I was proud to be his disciple.

And then there came his striking discovery of the third antigen in typhoid bacteria, the Vi-antigen. And from this in his hands grew the method of screening carriers through Vi-agglutination, a technique that saved so many lives and so much money.

In his last years he threw himself wholeheartedly into the work that became possible through Craigie's discovery of the adaptable phage, now known as 'phage-typing'. But it is idle to try and give a complete picture of this man's scientific work; I have merely touched on the main features.

Of my feelings of personal loss I cannot even begin to write. We were friends from 1928 onwards, and I never knew what I loved and admired most in him—his intellectual gifts, his tenacity in his work, his unlimited generosity, or his everlasting readiness to help one in one's problems. There was so much more in him, and he was so much to me.

DR. L. S. WILLIAMS

We regret to record the death of Dr. L. S. Williams, F.R.C.S. (Edin), Chief Medical Officer, New Consolidated Goldfields, Ltd., which took place in Johannesburg on 13 February. Dr. Williams was well known for his public work in the medico-social field, especially in the South African Red Cross Society, of which he was Chairman of the National Executive Committee, and in the South African National Tuberculosis Association (SANTA), of which he was Vice-Chairman. We hope to publish an In Memoriam article in the near future.

SELECT LIST OF RECENT ACCESSIONS TO THE MEDICAL LIBRARY, UNIVERSITY OF CAPE TOWN

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Faraday society. Chromatographic analysis. London, Gurney and Jackson, 1949.

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Grove, D. C. and W. A. Randall. Assay methods of antibiotics: a laboratory manual. New York, Medical encyclopaedia, 1955.

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Josiah Macy, Jr. foundation. *Administrative medicine. 3rd conference, Princeton, N.J.* 6, 7, 8, 1954. Transactions. New York, The Foundation, 1955.

Keet, A. D. jnr. The diagnosis of gastric mucosal prolapse with emphasis on the radiological features. s'Gravenhage, Excelsiors foto-offset, 1952.

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Nabarro, J. D. N. Biochemical investigations in diagnosis and treatment. London, Lewis, 1954.

Pugh, D. C. Roentgenologic diagnosis of diseases of bones. Baltimore, Williams and Wilkins, 1954.

Rajam, R. V. and P. N. Rangiah. Donovanosis. Geneva, W.H.O., 1954.

Rowe, N. L. and H. C. Killey. Fractures of the facial skeleton. Edinburgh, Livingstone, 1955.

Sapeika, N. Actions and uses of drugs. 4th edition. Cape Town, Balkema, 1955.

Shattuck, G. C. Tropical medicine at Harvard, 1909-1954. Boston, Harvard school of public health, 1954.

Spence, J. and others. A thousand families in Newcastle upon Tyne. Oxford U.P., for Nuffield foundation and Nuffield Provincial Hospital Trust, 1954.

Weinman, J. P. and H. H. Sicher. Bone and bones. 2nd edition. St. Louis, Mosby, 1955.

Winderen, L. and J. Zimmer. Cholesteatoma of the middle ear. Oslo, Rikshospitalet, Depts. of otolaryngology and radiology, 1954.

Worth, C. A. and B. Chavasse. Squint. 8th edition. London, Baillière, Tindall and Cox, 1950.

Wright, G. P. An introduction to pathology. 2nd edition, reprinted. London, Longmans Green, 1955.

New Periodicals

Acta medica orientalia; the Palestine and near East medical journal, v. 2, 1943 v. 7, no. 6, 1948, v. 11, 1952—Tel-Aviv, Palestine.

Acta oto-laryngologica, v. 44, fasc. 1, Jan/Feb. 1954—Stockholm, The Firm.

Acta paediatrica, v. 33, no. 1, 1945; v. 34, 1946; supplement 1, 1947; v. 44, no. 1, 1955—Uppsala, Almgqvist and Wiksell.

Advances in virus research, v. 1, 1953—New York, Academic press.

Archiv für Japanishe chirurgie, v. 221, no. 1, Nov. 1952—Kyoto, Chirurgische universitaetsklinik.

Biológia, Fasc. 6/7, 1947—Chile, Universidad de Chile.

British journal of haematology, v. 1, no. 1, Jan. 1955—Oxford, Blackwell.

Canadian journal of microbiology, v. 1, no. 1, Aug. 1954—Ottawa, National research council.

Central African journal of medicine, v. 1, no. 1, Jan. 1955—Salisbury, The Board.

Clinical chemistry, v. 1, no. 1, Feb. 1955—Baltimore, Hoeber.

Excerpta medica; section vii: Pediatrics, v. 1, 1947—v. 2, 1948; v. 3, no. 5, May 1949; v. 9, 1955—Amsterdam, Excerpta medica.

Experientia, v. XI, no. 1, 1955—Basel, Birkhäuser.

International journal of alcohol and alcoholism, v. 1, no. 1, 1955—Oxford, Blackwell.

Jaarboek van kankeronderzoek en kankerbestrijding in Nederland, no. 3, 1953—Amsterdam, Vereeniging het Nederlandsch kankerinstituut.

Japan medical association, Tokyo. Journal, v. 29, no. 7, April 1953—Tokyo, The Association.

Japanese safety forces medical journal, v. 1, no. 1, March, 1954—National safety medical association.

Journal of chronic diseases, v. 1, no. 1, 1955—St. Louis, Mosby.

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St. Francis Hospital and sanitorium, Roslyn, N.Y. Bulletin v. 11, no. 1, Jan. 1954—Roslyn, N.Y., St. Francis hospital and sanatorium for cardiac children.

Serbian archives of medicine, v. 81, no. 11, 1953—Belgrade, Serbian medical association.

South African journal of laboratory and clinical medicine, v. 1, no. 1, March 1955—Cape Town, South African medical association.

South African journal of medical laboratory technology, v. 1, no. 1, 1955—Durban, Society of medical laboratory technologists of S.A.

United nations. *World health organization, Geneva.* The work of WHO, 1951—Geneva, The Organization.

PASSING EVENTS : IN DIE VERBYGAAN

Cecil John Adams Memorial Trust Travelling Fellowships. Awards of £750 each for this year have been made to the following:

(1) Dr. G. S. M. Botha of Cape Town, now doing research work at the Queen Elizabeth Hospital, Birmingham. (2) Dr. C. N. Barnard of Pinelands, Cape, who is doing research work at the University of Minnesota, Minneapolis. (3) Dr. D. M. Krikler of Cape Town, who will be doing research work at the Central Middelsex Hospital, London, and/or the Lakey Clinic, Boston, Massachusetts. (4) Dr. T. L. Sarkin of Sea Point, Cape, who intends doing research work at the Royal College of Surgeons, England, and various Orthopaedic Centres in the United Kingdom. (5) Dr. V. Botoulas of Hillbrow, Johannesburg, who intends doing research work at the Postgraduate Medical School, Hammersmith, London, and the National Heart Hospital, London.

This trust was established by the late Mr. Arthur Edward Adams, of Cape Town, in memory of his son Cecil John Adams, and is administered by the South African Association. The income is paid annually to medical men for research work overseas.

* * *

Nuffield Dominion Travelling Fellowships for 1957. Applications are invited *not later than* 30 *April*, 1956, for the following senior fellowships offered to South African nationals by the Nuffield Foundation for the year 1957: One fellowship in the Natural Sciences. One fellowship in Medicine. Two fellowships in the Humanities. An additional fellowship may be awarded in any one of these fields.

Applicants should normally be between 25 and 35 years of age, have high intellectual and personal qualities, and have shown capacity of an unusual order for advancing knowledge and education in some branch of their fields. They must undertake to return to South Africa as soon as possible after the termination of their fellowships.

Copies of the regulations and application forms may be obtained *by university applicants* from Registrars of their universities, and *by others* from the Honorary Secretary, Nuffield Foundation, South African Advisory Committee, c/o University of the Witwatersrand, Johannesburg.

* * *

Cape Western Sub-group of the Radiological Society of South Africa. The annual general meeting of the Cape Western Subgroup was held in Cape Town on 6 February 1956, when the following officers were elected: Chairman Dr. Adolph Meyer, Vice-Chairman Dr. H. J. B. Muller, Hon. Secretary Dr. K. V. O. Gunn.

* * *

Dr. Louis Meyerson, M.B., B.Ch., D.O., R.C.P. & S. (Eng.), has commenced practice as an opthalmic surgeon at 606-608 Clinical Centre, 5 Wanderers Street (between Bree and Plein Streets), Johannesburg. Telephones: Rooms 23-7785, Residence 42-1049.

The Fourth International Congress on Diseases of the Chest, sponsored by the Council on International Affairs of the American College of Chest Physicians, will be held in Cologne, Germany, on 19-23 August 1956. The Congress is being officially sponsored by the German Federal Government, and Chancellor Konrad Adenauer is serving as the Honorary President. The Foreign Office of the German Government is extending official invitations to all governments with which they have diplomatic relations, requesting that delegates be designated to attend the Congress. The Medical Association of South Africa has been invited to appoint an official delegate.

Information concerning the Congress may be obtained from the Congress President, Prof. Gerhard Domagk. c/o Executive Offices of the American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, USA.

* * *

Doctors' Room at the Medical Library, Medical School, Observatory, Cape Town. The Council of the Cape Western Branch has approved of an appeal for funds for the furnishing of the attractive Doctors' Room at the new Medical Library. This room has been specially designed for use by our town and country members. It provides for a restful atmosphere where the busy practitioner may research for a little while in comfort. As no funds are available for its furnishing, it is felt that members should be given the opportunity to provide it themselves.

The Honorary Librarian has arranged for specially designed furniture in imbuia (stinkwood is not available) by one of our leading firms. The total cost will be in the neighbourhood of £400. This year's postgraduate class has already donated £40.

Donors may present the larger pieces, in which case suitable brass plates acknowledging the gifts will be attached. Cheques or indications of the actual donation of items should be made out to the Medical Library and sent to the Hon. Librarian, Dr. Harold O. Hofmeyr, 148 St. George's Street, Cape Town.

BOOK REVIEWS : BOEKRESENSIES

A CYTOLOGIST ON CANCER

Cancer Cells. By E. V. Cowdry. Pp. 677 + xvi, with 137 illustrations. £6 16s. 0d. Philadelphia and London: W. B. Saunders Company. 1955.

Contents: 1. Cancers Defined. 2. Malignant Growth of Cancer Cells. 3. Cytoplasm of Normal and Malignant Cells Compared. 4. Nuclei and Division of Normal and Malignant Cells Compared. 5. Chemical Properties of Cancerous Tissues. 6. Occurrence of Cancers in Animals and Plants. 7. Agents Causing Cells to Become Malignant. 8. Single Trauma Cancers. 9. Viruses as Cancer Agents. 10. Mutations as Cancer Producers. 11. Cellular Susceptibility to Cancer. 12. Heredity and Susceptibility to Cancer. 13. Age and Sex Susceptibility of Cells to Cancer. 14. Latency in Cancer Development. 15. Modifying Factors in Cancer Development. 16. Geographic Frequency of Different Types of Cancer. 17. Cancer Prevention. 18. Cancer Diagnosis and Treatment. 19. Unexplained Disappearance and Changes in Malignancy of Cancer Cells. 20. Cancer Research. Appendix. Bibliography. Index.

Cowdry has a striking record in medical bibliography as an editor of comprehensive works, and those who know and value General Cytology, Special Cytology, or Problems of Ageing, will be interested to note that he has attempted this vast field on his own. The list of chapters will indicate how widely he has interpreted his title—he ranges from pure cytology to cancer treatment, demography and the administration of cancer research funds.

This subject, so often dealt with by pathologists and surgeons, has long needed reviewing by a cytologist, and the work can be read with much profit by both expert and beginner, who will find it well within his comprehension. The roots of many terms are given in the text—perhaps too didactically, for it is distracting to encounter a digression explaining the well-known connection between the term 'muscle' and 'mouse', or to find 'spontaneous' explained in two places in the text. Such defects have made the writing a trifle ingenuous in parts.

It is nevertheless refreshing nowadays, when the prevalent tendency is to employ so many authors that a theme is buried in detail and conflicting views, to find such a vast work attempted by a single author; yet one must regretfully conclude, as Cowdry admits, that the subject after all, was too big for one man. It might have been possible to divide this book among 3 or 4 with advantage, but when one attempts a labour of this size, and manages to be up-to-date in the references, it is impossible to provide those benefits which would justify single authorship, viz. the considered opinion of an expert in the field, rather than the mechanical review of recent articles. In parts, it is regretted, this book tends towards the latter. Fibiger's claim to have induced stomach cancer by nematode infestation for example is mentioned, also that others have doubted this, but the author does not take sides. In other sections gaps occur; e.g. there is no mention of J. R. Baker's work on the lipochondria in the discussion of the Golgi apparatus. Some errors are hard to explain; thus head abnormalities in the sperm are quoted as an indication of impotency instead of sterility. The statement had no bearing on the subject under consideration, and the error simply annoys the medical reader.

There is a hint of the dictaphone in this work—errors, clichés and misprints are unusually numerous and it lacks those scholarly standards that unfortunately also unduly defer the publication date.

Despite this criticism, which is one of detail and hastiness of style, one must emphasize that the extent both of the author's knowledge and its range is immense. About 2,400 references are quoted, not all dealing with cancer, but much is included that one would otherwise have great difficulty in finding. His description of modern cytology in the light of electron microscopical studies is particularly good. There are numerous illustrations, mostly from published work by courtesy of the authors. It is probably the very quality of this work and the experience of its author that makes what after all are minor defects the more detectable. As a general detailed introduction to many aspects of this subject, it is unrivalled, and a tribute to the immense experience of the author. It is a work of reference to which I shall turn again and again.

On the subject of the support of cancer research Cowdry's views deserve serious consideration, more especially his conclusion that the present trend towards annual grants, after long and detailed reports, is an unfortunate development, and permanent endowment would be far more efficient.

The difficult question remains, what should have been left out? Even the chapter on treatment, obviously out of place and too briefly dealt with to be of much use, has sections one would be sorry to miss. Still the cost of this book is too high and much could have been excluded without serious loss, including some of the illustrations, by more careful editorial pruning. Who, one wonders, would edit Cowdry?

A.G.O.

TROPICAL INFANT NUTRITION

Infant Nutrition in the Subtropics and Tropics. By D. B. Jelliffe, M.D., M.R.C.P., D.C.H., D.T.M. & H. Pp. 237, with illustrations. £1 5s. 0d. Geneva: World Health Organization. 1955.

Contents: 1. Evolution of Infant Feeding in the Western World. 2. Present Infant-Feeding Practices in the Subtropics and Tropics. 3. Present Status of Nutritional Disease Among Infants in the Subtropics and Tropics. 4. Methods of Improving Infant Feeding in the Subtropics and Tropics. 5. Prevention of Kwashiokor. 6. Nutrition Education in the Maternal and Child Health (MCH) Centre. Acknowledgements. Illustrations. References. Index.

The high infant mortality and morbidity of the tropics, subtropics and other less privileged parts of the world is undoubtedly linked with poor nutrition. In this monograph it is rightly pointed out that, to effect a general improvement of nutrition in population groups, it is necessary to have a better knowledge of existing feeding practices in those groups.

The author begins with a brief and interesting review of infant feeding through the centuries in the Western world. He goes on to a description of present-day methods in a large number of countries in Africa, the Middle East and Asia, which he himself has visited. In the sections on prevention and education he stresses that to modify ideas and so improve the practices of people in nourishing themselves and their children, is no easy task. He points out that the Maternal and Child Health Centre and its workers need a much more imaginative approach than has been usual in the past. Several useful suggestions are made on how to work with the people and how to make them enthusiastic about nutrition education.

The middle section of the book is concerned with an up-to-date, brief description of specific nutritional disorders, their differentiation, treatment and prevention. In particular, nutritional anaemia, avitaminosis A, infantile beri-beri, rickets, kwashiorkor, and marasmus, are extremely well clarified. The prevention of kwashiorkor is dealt with at length and there are some good coloured prints of the condition. A comprehensive bibliography of 313 references is included.

This is a very creditable World Health Organization publication and can be recommended to all those who have to deal with the problems with which it is concerned.

J.H.

ADVANCES IN INTERNAL MEDICINE

Advances in Internal Medicine. Volume II. Edited by William Dock, M.D. and I. Snapper, M.D. Pp. 311, with illustrations. $8.50. Chicago: Year Book Publishers, Inc. 1955.

Contents: 1. Pathogenesis and Treatment of Renal Lithiasis—Newer Concepts. 2. Hepatic Coma. 3. The Pathophysiology of the Pancreas. 4. Clinical Significance of Serum Mucoproteins. 5. Advances in Physiology of Clinical Disorders of the Adrenal Cortex. 6. Diseases of the Pericardium. 7. The Nephrotic Syndrome. 8. Applied Pulmonary Physiology. Indices.

It is the policy of the editors of Advances in Internal Medicine to present reviews on from 8 to 10 subjects per volume. These reviews are usually by writers in the particular fields who have in most cases themselves made contributions to knowledge in them. So we have in this volume C. S. Davidson writing on Hepatic Coma. The role of ammonia in the production of the neurological manifestations of cholaemia and the question whether we should give or withhold protein and amino acids in such cases, or perhaps give glutamic acid, are the substance of the more recent studies.

Butt discusses the pathogenesis and treatment of renal lithiasis and presents the case for subcutaneous hyaluronidase. He quotes a recent report that oral glucuronic acid, which is a break-down product of hyaluronic acid, has a similar effect. ACTH and cortisone have the effect of decreasing urinary glucuronic acid. Butt does not mention the known effects of salicylates in this connection and the recent work which showed beneficial results in cases of urinary calculus.

Dreiling and Janowitz write on the Pathophysiology of the Pancreas. They discuss what we now know to be the secretin complex with its 5 hormones. They review the difficult subjects of the pathogenesis and diagnosis of pancreatic disease. They omit any mention of steroid therapy in pancreatitis.

Jailer reviews the clinical disorders of the adrenal cortex and surprisingly says nothing of aldosteronism.

McKusick and Harvey deal with diseases of the pericardium. It is worth mentioning that the neck veins often pulsate in cases of pericardial effusion, and so may the liver. The work of Isaacs *et al.* is quoted which shows that constrictive pericarditis constricts not the great veins but the chambers of the heart. McKusick himself has described an instance of 'constrictive endocarditis'.

Squire writes on the nephrotic syndrome, and discusses such aspects of the problem as the source of the urinary protein and the mechanism of oedema formation. He concludes that for the present the graded-pore theory of the glomerulus seems most readily to account for the known facts.

Greenspan deals with the subject of serum mucoproteins and their changes in disease—hepatobiliary disease, rheumatic and collagen diseases, etc.

Stone reviews the subject of applied pulmonary physiology. Elastic and viscous forces are discussed and the main topic is naturally that of emphysema.

F.F.

CANCER OF THE BREAST

Breast Cancer and Its Diagnosis and Treatment. First Edition. By Edward F. Lewison, B.S., M.D., F.A.C.S. Pp. 478 + xii with 181 illustrations and 4 coloured plates. 114s. 0d. London: Baillière, Tindall & Cox Ltd. 1955.

Contents: 1. The History of Breast Cancer and its Treatment. 2. The Surgical Anatomy of the Breast. 3. Physiology of the Breast. 4. Experimental Aspects of Mammary Cancer in Mice. 5. The Pathology of Cancer of the Breast. 6. The Relationship Between Benign Breast Disease and Cancer. 7. The Psychological Aspects of Breast Cancer. 8. Diagnosis. 9. Breast Biopsy. 10. A Review of the Surgical Treatment of Breast Cancer. 11. The Surgical Treatment of Breast Cancer. 12. Extended Radical Mastectomy. 13. Postoperative Care. 14. Prognosis. 15. Radiotherapy of Breast Cancer. 16. Hormonal Alterations in the Palliative Treatment of Breast Cancer. 17. Breast Cancer and Pregnancy or Lactation. 18. Self-Examination of the Breast. 19. The Statistics of Breast Cancer. 20. Cancer of the Breast in the Male. Index.

The Greek scholar, Porson, wrote that German philosophers dive deeper and come up muddier than their colleagues. With equal justice this might be said of writers on cancer of the breast. Surely, one exclaims, the subject does not warrant the publication of another book, and one costing £5 14s. at that. The dismal facts are known, the side-issues have been sufficiently dealt with in numberless articles, and the welter of statistics has been churned over, digested and regurgitated *ad nauseam*.

And yet no-one who reads this remarkable volume can fail to be impressed. As the list of contents shows, almost every conceivable facet of the disease is examined and almost every problem illuminated in the light of such knowledge as we possess.

In the last analysis what both doctor and patient want to know is the diagnosis, treatment and prognosis of cancer of the breast. And, of these, diagnosis is the simplest issue, treatment and prognosis the most complicated. We teach and are taught that the presence or absence of axillary gland metastases is of paramount importance in prognosis. But clinical assessment of axillary involvement is subject to gross error. Haagensen is quoted as showing that 44% of his cases had axillary metastases on microscopic study when the examiner had regarded the axilla as clinically innocent, and 15% were negative microscopically where clinical examination had been positive. Microscopic examination of the axillary glands, to be useful, means a study of all glands in serial section. This is time-consuming and expensive, with the result that most pathological reports on excised specimens are incomplete. Therefore if prognosis is to depend on knowledge of the state of the axillary glands a source of significant error arises in a majority of cases.

Even more alarming is the possible implication of internal mammary and supraclavicular glands. Handley and Dahl-Iversen

have made clinical staging even more difficult as a result of their work, so that in the words of the author of this book 'with true humility we must confess that despite prompt and adequate treatment we do not know precisely which individual patient will be favoured by fortune and granted a long survival'. But when we come to define 'prompt and adequate treatment' we are once again on uncharted seas. On the one hand are the therapeutic nihilists who tell us gloomily that no method available today influences the final result, on the other the radicals who want the breast removed the axilla cleared, the chest wall opened to extirpate the internal mammary glands and the supraclavicular nodes dissected in continuity—a formidable programme which has gained few adherents.

This book, reflecting Johns Hopkins teaching and tradition, favours radical mastectomy with radiotherapy for operable cases and radiotherapy for others. It rejects the McWhirter approach (see in this connection a valuable statement by Ackerman on the Edinburgh plan). There is a detailed discussion of the role of radiotherapy and hormone treatment, with much practical and useful information on the handling of patients.

All in all, this work is a massive contribution to the subject. It is a pity that every now and then the principal writer breaks into Bombast—Babu English. What, for instance, does one do except squirm in discomfort, when one comes across sentences such as the following? '. . . . in a patient with a large fungating tumour where there are firm fixed regional nodes in which metastases have undoubtedly passed through the shadow of the valley of the axilla' '. . . . is the armour of education adequate to arouse curiosity in the common danger signals of breast cancer'. And what on earth does this mean? '. . . . the epic of past experience has grimly indicated that cancer of the breast has never been at a loss for occasions and it is not unreasonable at present to forecast its continued malevolence'. And what has Heneage Ogilvie done to deserve being called 'the kaleidoscopic Sir Heneage'? Bombast—Babu dominates surgical writing to-day. Its disciples never discuss the treatment of piles. They write an evaluation of the modalities of treatment in ano-cutaneous varicosities. They adorn their shopworn prose with itsy-bitsy little tags from Osler, who was a bore anyhow, and they add pounds to the weight of surgical journals. A pox on them!

It is only fair to add, however, that chapters of this book are models of lucidity and that, despite these minor irritations, this reviewer will cherish his copy.

G.S.

BOOKS RECEIVED : BOEKE ONTVANG

The Diagnosis and Management of Urological Cases. By Bruce W. T. Pender, M.B., B.S., F.R.C.S.; James O. Robinson, M.A. (Cantab.), M.Chir., F.R.C.S.; Foreword by Sir W. Heneage Ogilvie, K.B.E., M.A., M.D., M.Ch., F.R.C.S. Pp. 170, with illustrations. 21s. London: Baillière Tindall and Cox. 1955.

Neural Control of the Pituitary Gland. By G. W. Harris, F.R.S., Sc.D., M.D. Pp. 298, with illustrations. 30s. London: Edward Arnold (Publishers) Ltd. 1955.

Il sottosesso nei Popoli Senza Tonsille. By I. e G. Calderoli. Pp. 155. Bergamo: Tipografia Orfanotrofio Maschile. 1954.

Arthroplasty. By St. J. D. Buxton, M.B., B.S., F.R.C.S. Pp. 126, with illustrations. 30s. London: Pitman Medical Publishing Company Ltd.

An Atlas of Bone-Marrow Pathology. By M. C. G. Israëls, M.Sc., M.D., F.R.C.P. Second Edition. Pp. 85, with illustrations. 30s. net. London: William Heinemann Medical Books Ltd.

Year Book of Radiology. (1955-1956 Year Book Series.) By John Floyd Holt, M.D.; Fred Jenner Hodges, M.D.; Harold W. Jacox, M.D. and Morton M. Kligerman, M.D. Pp. 413, with illustrations. Chicago: The Year Book Publishers, Ind.

Blood Supply and Anatomy of the Upper Abdominal Organs. By Nicholas A. Michels, M.A., D.Sc. Pp. 581, with illustrations. £8 net. London: Pitman Medical Publishing Co. Ltd.

Atlas of World Distribution of Spirochetal Diseases 2. *Relapsing Fevers* (*Louse-Borne and Tick-Borne*) (*Plate* 16). Department of Medical Geography: New York.

Atlas of World Distribution of Spirochetal Diseases 3. *Leptospiroses* (*Plate* 17). New York: Department of Medical Geography.

CORRESPONDENCE : BRIEWERUBRIEK

THE BENEFIT SOCIETY SYSTEM

To the Editor: In the issue of your *Journal* of 4 February 1956 there appears a report[1] by a sub-Committee appointed by Federal Council of the South African Medical Association to report on the economics of medical practice, and nearly a third of this report is devoted to critical, and unfavourable, comment on the medical benefit society system, and in particular on the methods and practices of the Mines Benefit Society. This part of the report presents so distorted a picture of the case of the Benefit Society *versus* the Medical Profession that I feel bound to ask you to accord me some space in your columns to enable me to dispel some of the illusions it may well create.

At first reading the report gives so harrowing an impression of the lot of the medical officer under the dominance of a benefit society, and of my Society in particular, that one could almost imagine that the days of Simon Legree had come again, with my Society filling the role of the brutal taskmaster of helpless practitioners who have been kidnapped and enslaved, oppressed and starved. In fact, a practitioner is under no compulsion to seek appointment as a medical officer in the first place and if, having sought and obtained appointment, he finds the conditions, whether of remuneration or of service, unsatisfactory he can at any time relinquish it at short notice. Does not the fact that the Society continues to receive more requests than it can satisfy for appointment as panel doctor, that the number of resignations is so small as to be negligible, and that in none of these is dissatisfaction with remuneration or other conditions of service assigned as the cause, indicate that there is nothing gravely wrong with those conditions?

An explanation of this phenomenon seems to be offered in paragraph 41 of the Report. It is there said that 'the acceptance of lower fees by the profession amounts to a subsidy granted to the lower-income groups in order that they may protect themselves at a reasonable rate of subscription against the crippling costs of serious illness.' If I read this aright the implication is that practitioners who seek appointment as medical officers do so at the urge of humanitarian motives impelling them, at whatever financial sacrifice, to give service to the unfortunate employees of the mining industry; or if it does not mean that, what does it mean? Now I have very good reason to know that very many medical men are ready at all times to give *pro deo* service in case of real need, but I do feel some doubt whether the humanitarian urge is so widely and so strongly felt, even in the medical profession, as to induce its members to queue up for appointment to underpaid and overworked posts in order that they may thus be of service to their needy fellow men.

Paragraph 31 of the Report is a quite unfair and unjustified presentment of the attitude of the managing bodies of this Society, and probably also of that of other such societies. Doubtless there are committee members who find it difficult to see and appreciate any side of a case other than their own, but it may be suggested, with all respect, that such as they are to be found on the committees of all organizations, even perhaps on those of the Medical Association itself. It can certainly be said that that is not the outlook of the majority on the management committees of this Society; that that is so is shown clearly by the action of the Society in granting the increases to which the report, even if in terms of some disparagement, refers.

During the 14 years of my association with the Society its relations with its medical officers, both specialists and general practitioners,

have in general been, and still remain, harmonious. The great body of members is appreciative of the services that they receive at their doctors' hands, and it is believed that the doctors, on the other hand, are conscious that the Society strives to be just and fair in its dealings with them. Medical officers, of course, would like to receive higher remuneration; that is natural. It is no less natural that members should dislike having to pay the larger subscription which an increase in medical officers' remuneration would entail. While such conflicts of interest are inevitable they can be resolved as they have in the past been resolved, but by negotiation and not by vilification. I do not propose here to deal further with the question of the adequacy of present remuneration. It is a subject that has many facets and it would be out of place here to enter upon, or to continue, a discussion of it. I do want to say, however, that it is much to be deplored that the harmony now prevailing should be disrupted by the publication of such a report as this, the object of which appears clearly to be to foment groundless discontent.

In paragraph 32 of the Report it is said that a careful study has been made by lay bodies of the working of the Medical Association for the purpose of discovering its weaknesses. This Society has certainly attempted no such study. To it the workings of the Association remain as profound a mystery as they are doubtless to most of its members. But if it were true of the Society it would hardly be matter of reproach by a committee of the Association which has been at pains and expense to gain a knowledge of the internal transactions of the Society.

Generally, the report is critical of the 'medical benefit society system'. Now there are, of course, many weaknesses and defects in the system, but it still remains the most effective means of protection against the crippling costs of serious illness, the necessity for which seems to be conceded by the reporting committee, and it is doubtless for that reason that the National Health Service of Great Britain and a projected similar service for the Union have been cast in this form. Some of the weaknesses could be improved, even if not eliminated, by the doctors themselves as, for instance, the undue demand upon a doctor's time by those who do not really need his services. It is only the doctor who can tell when this occurs; it is only the doctor who can tell the 'pseudo-patient' that he is wasting his time, and tell him in terms calculated to discourage a repetition. Many doctors, it is believed, do this with good results, but others say that they cannot do it for fear that the member may seek transfer to another panel—an attitude that suggests that even a time-wasting and exigent patient is profitable at the present rate of capitation fee.

Then there is the question of over-large panels. That is clearly a matter to be remedied by the panel doctor himself. It is widely known that attempts made by the Society from time to time to limit the size of panels have met with the strongest resistance by medical officers. It seems to be suggested, though not stated, that large panels result from the insufficiency of the capitation fee; but is it seriously suggested that if the capitation fee were increased by, say, 50% panel-doctors with large panels would immediately ask for the removal from their panels of a proportionate number of members?

On the other hand there is a proposal that the Society itself could do something to curb excessive demand upon its doctors, and upon medicines, by prescribing payment by members of fees of nominal amount on consultation or of part of the cost of medicines. Now these proposals certainly deserve full examination and consideration and though it is much to be feared that, attractive as they may at first sight appear, they will be found to be impracticable, that fear should not stand in the way of their fullest exploration.

There are many other averments and innuendoes in the report that I could wish to traverse, but the doing of it would take up much of my time (which I could spare) and much of your space (which I can hardly ask you to give).

Let me conclude by saying that early in this letter I spoke of 'the case of the Benefit Society *versus* the Medical Profession'. That is a complete misdescription of the question in issue and was used only because that seems to be the way in which the report under discussion presents it. It should be corrected by substituting *cum* for *versus*, for the common objective of the Society and the profession is, or should be, the provision of needed health services on terms which are just and equitable both to the member and to the medical officer. That objective can be obtained by cooperation, but not by antagonism. Finally let me say that though the benefit society system brings with it great advantages to the medical profession it must be remembered that that is not the main purpose of its existence.

S. Maynard Page
Chairman

Mines Benefit Society
P.O. Box 8603
Johannesburg
10 February 1956

1. Report (1956): *Contract Practice with particular reference to Conditions existing in the Transvaal.* S. Afr. Med. J., **30**, 114.

CHEMICAL AND BACTERIOLOGICAL INVESTIGATIONS

To the Editor: A letter from Dr. T. H. Crouch[1] in your *Journal* of 14 January, 1956 shows the desirability of making clear to practitioners the procedure to be followed in cases of suspected gastro-intestinal poisoning. Dr. Crouch remarks that the specimen in question was forwarded by him to the Institute for Medical Research but it is clear from his letter that he suspected chemical and not bacterial food poisoning. Specimens of this nature should be forwarded directly to the Government Chemical Analytical Laboratory and not to the Institute for Medical Research as the latter does not carry out toxicological analyses. If, on the other hand, bacterial food poisoning is suspected the Institute for Medical Research carries out investigations on specimens submitted on behalf of the Secretary of Health. For toxicological analyses the Government Chemical Analytical Laboratory requires authorization by the Public Prosecutor and it was for this reason that the Government Laboratory correctly requested Dr. Crouch to comply with the normal procedure. In the case of bacterial food poisoning no authority by the Public Prosecutor is required as such investigations, unlike toxicological analyses, seldom, if ever carry medico-legal implications.

It is unfortunate that unless there are possible criminal implications, no Public Laboratory mechanism exists in South Africa for investigation of cases of suspected chemical poisoning—not even when such an investigation would be of medical importance.

Allow me to thank you for the courtesy of your columns.

J. F. Murray
Medical Superintendent
Routine Diagnostic Department.

South African Institute for Medical Research
P.O. Box 1038
Johannesburg
2 February 1956

[Should a doctor suspect poisoning and wish to have a specimen chemically examined he should apply to the Magistrate or Police if criminal poisoning is suspected, in order to get the necessary authority for analysis by the Government Chemical Analytical Laboratory. If criminal poisoning is not suspected but the matter is of public-health interest he should apply to the Secretary for Health, Pretoria, who may give such an authority. In lead poisoning or poisoning by insecticides, both of which are cumpulsorily notifiable under the Public Health Act, the Department of Health would bear the cost of investigations, including chemical analyses. —*Editor.*]

1. Crouch, T. H. (1956): S. Afr. Med. J., **30**, 48.

'MONGOLISM' IN THE BANTU

To the Editor: I am grateful for the interest taken by Dr. Ben Epstein[1] and Dr. P. Klenerman[2] in my article[3] on this subject. (See their letters published in the *Journal* of 3 and 10 December 1955.)

The Zwelitsha Health Centre was not a small one; a doctor, 2 medical aides, 6 nurses, and 6 health assistants provided a curative-preventive medical service, which was intensive and efficient, to about 12,000 Africans. Babies less than 2 years of age numbering 1,896 attended the health centre for the first time in the calendar year 1952. Others had attended also in 1950-51. The child-welfare clinic had 9,000 attendances in that year, and there were, in addition, 3,000 attendances for morbidity. In the 2-15 year age-group there were 5,824 attendances (all for morbidity) including 1,573 first attenders. I am convinced that the major proportion of children resident in the area did attend the health

centre during the year 1952, and this applies even more to babies in their first and second years. The sick almost certainly attended (there were few other medical facilities) and one expects that mongols would be included with the sick, rather than with the healthy group who do not seek medical aid.

In Thaba 'Nchu in 1949, 647 babies less than 2 years of age attended the health centre, and about 2,000 children aged 2-18 years.

No case of mongolism was found in Thaba 'Nchu, and the case which I reported in the *Journal* of 5 November 1955 was the only one identified in the larger health centre. Other observers confirmed my impression that the condition was rare. I shall, however, agree with Dr. Eptsein that my sample should be larger for the evidence to be conclusive.

The fact that cases are more frequently seen in the urban areas of Durban and Johannesburg may be due to (1) the larger population, (2) visitors (not local residents) who have come from far and wide to the experts in the large hospitals, (3) admixture of new strains in the urban environment, or (4) an environmental factor, e.g. infection during early pregnancy, or nutritional, which may be operative to a greater extent in urban areas.

Further investigation is required, and if my article has been a stimulus I am more than content.

B. J. Kaplan

P.O. Box 267
Bethlehem
10 February 1956

1. Epstein, B. (1955): S. Afr. Med. J., **29**, 1155.
2. Klenerman, P. (1955): *Ibid.*, **29**, 1183.
3. Kaplan, B. J. (1955): *Ibid.*, **29**, 1041.

EXPERIENCE WITH MITRAL VALVOTOMY AT GROOTE SCHUUR HOSPITAL

To the Editor: Mr. Adler[1] has clearly failed to understand our article[2] even after reading it for the second time. He has not only taken some important views of ours from their context but has committed the double sin of misquoting them. This reply is necessary as he may have caused others to become similarly confused.

In no place in our article did we offer a definition of a wide opening of the valve. I try to restore the valve to its normal size if at all possible and only if it is safe. *The* 3 *sq. cm. standard* is used by me for two good reasons. Surgeons are able to measure with their fingers a valve opening of this size with some degree of accuracy. I have used the term *over* 3 *sq. cm.* when the valve opening is obviously larger and can be measured only approximately. In many cases the valve is opened to an estimated size of 6 sq. cm. or more. The 3 sq. cm. standard is used furthermore because numerous physiological estimations have indicated that a valve of this size is adequate for circulation in an adult.

I consider that it is unwise to aim for a full valvotomy with the use of a knife where there is difficulty. *Safe partial valvotomy has been the method of choice rather than a full valvotomy with its attendant dangers of mitral regurgitation.* In the ideal case valvotomy takes a few seconds with minimal disturbance. The valve can be split with the finger alone, the final size being more than 3 sq. cm. and perhaps as much as 7 sq. cm.

Mr. Adler is also surprised that I used the knife in only 3 of the last 48 cases of the series. He thinks that this is out of date and not in keeping with current thoracic surgical experience. I found that it was necessary to use the knife in almost 50% of my early cases. *I found the knife was required less frequently as I gained experience and as I modified the technique.* Claude S. Beck wrote in November 1954, 'During the past 15 months I have used this method of enlarging the orifice of the stenotic mitral valve on about 200 cases. After I began to use the method I found that it was not necessary to cut the valve'. Mr. Adler may have a description of this technique and a long bibliography from me by letter if he so wishes, as I do not wish to take up readers' time.

I am well aware of sub-valvular stenosis. No attempt was made to separate adherent chordae during the exploration of the left ventricle in this series of cases. In many cases it is difficult to palpate the subvalvular region properly unless an ungloved finger is used. There is great danger of tearing chordae during the separation of adherent parts.

We have said that we must wait and see whether stenosis will recur if the valve is not opened completely. It may occur in spite of a complete opening. Every observer has reported the presence of microscopically active rheumatic lesions in the walls of the atrial appendages removed at operation. One competent group of workers stated, 'We do not yet know whether the operatio .s . . . . will be of permanent value or . . . . only set the clock back for these patients by a few years'.

The test of adequacy of the operation has been measured in all cases by the careful follow-up of my physician colleagues. Mr. Adler states that in 50 cases in 1955 he used the finger and knife in 15 cases, and the knife only in 14 cases. Some form of instrumental valvotomy was done in 29 of the 50 cases. He also states that he fully agrees that not only is mitral regurgitation more likely to result from instrumental valvotomy, but also that regurgitation surgically induced is dangerous. I agree with him that the publication of results in the surviving cases after a further 5 years will prove instructive.

Walter L. Phillips

Cape Town
14 February 1956

1. Adler, D. (1956): S. Afr. Med. J., **30**, 155 (11 February).
2. Schrire, V., Vogelpoel, L., Phillips, W. and Nellen, M. (1955): *Ibid.*, **29**, 1108 (26 November).

RADIOTHERAPY IN CANCER OF THE BLADDER

To the Editor: Dr. Shulman,[1] in his letter in your issue of 11 February 1956, appears to have missed the point in my letter.[2] What irked me in the report by Dr. R. Paterson[3] was that, *in this article*, there were no data regarding the type of cancer he was treating, and, as such, the figures he gave were, to my way of thinking, useless as means of evaluating his results.

As regards the question of adequate transurethral biopsy of the tumour, on the basis of the precepts laid down by Jewett, I must reserve the right to disagree with Dr. Shulman regarding the extent to which this has been practised in Great Britain. Whilst by no means negating the great value that the various forms of radiotherapy have in the treatment of cancer of the bladder, I think Dr. Shulman would find few urologists who would agree with him that partial cystectomy is an obsolete operation, if carried out according to the tenets laid down by Jewett.

Until the results of radiotherapy in the treatment of carcinoma of the bladder, have been assessed along the lines I have just mentioned, I shall certainly be unable to draw any conclusions from the figures, no matter how excellent the physicians or the Institutions where they work are reputed to be.

J. N. de Klerk

623/4 Groote Kerk Buildings,
Adderley Street
Cape Town
13 February 1956

1. Shulman, I. M. (1956): S. Afr. Med. J., **30**, 156.
2. De Klerk, J. N. (1956): *Ibid.*, **30**, 24 (7 January).
3. Paterson, R. (1955): *Ibid.*, **29**, 1215 (24 December).

RELAXANTS IN ANAESTHESIA

To the Editor: I have been interested in the correspondence which your editorial[1] of 19 November 1955 has evoked. I wonder whether your various correspondents would care to comment upon the report by Dr. Michael Johnstone (M.D., F.F.A.R.C.S.) of severe cardiovascular collapse following the intravenous injection of suxamethonium (Scoline) in four fit young male patients undergoing minor operations? This report appears in an article entitled 'Relaxants and the Human Cardio-vascular System' in *Anaesthesia* (the Journal of the Association of Anaesthetists of Great Britain and Ireland) on pages 122 to 138 of volume 10 (April 1955). Details of these four patients are given by Dr. Johnstone (who is attached to the University Department of Anaesthetics of the Royal Infirmary, Manchester, England) on page 128 of his article, which is concerned with E.C.G. changes that develop when a variety of relaxants are used.

C. S. Jones

Department of Anaesthesia
University of Cape Town
14 February 1956

1. Editorial (1955): S. Afr. Med. J., **29**, 1084.